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Volume I

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16. ABSTRACT

On October 31, 1984, EPA published a notice in the Federal Register withdrawing proposed standards for radionuclide emissions from four sources: 1) DOE facilities, 2) NRC-licensed facilities and non-DOE Federal facilities, 3) underground uranium mines and 4) elemental phosphorus plants. This Background Information Document supports the Agency's final actions on radionuclides. Volume I is an integrated risk assessment. It addresses historical and current regulatory programs and strategies, hazard identifications (health effects), radionuclide emissions, reduction of dose and risk, movement of radionuclides through environmental pathways, radiation dosimetry, and estimating the risk of health effects resulting from radionuclide air emissions. Volume II examines the source categories and presents the following information for each category: a general description of the source category, a brief description of the processes that lead to the emissions of radionuclides into air, a summary of emissions data, and estimates of the radiation doses and health risks to both individuals and populations.

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(INTEGRATED RISK ASSESSMENT)

FINAL RULES FOR RADIONUCLIDES

VOLUME I

October 22, 1984

U.S. Environmental Protection Agency
Office of Radiation Programs
Washington, D.C. 20460

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CHAPTER 1: INTRODUCTION

1.1 History of Standards Development

In 1977, Congress amended the Clean Air Act (the Act) to address airborne emissions of radioactive materials. Before 1977, these emissions were either regulated under the Atomic Energy Act or unregulated. Section 122 of the Act required the Administrator of the U.S. Environmental Protection Agency (EPA), after providing public notice and opportunity for public hearings (44 FR 21704, April 11, 1979), to determine whether emissions of radioactive pollutants cause or contribute to air pollution that may reasonably be expected to endanger public health. On December 27, 1979, EPA published a notice in the Federal Register listing radionuclides as hazardous air pollutants under Section 112 of the Act (44 FR 76738, December 27, 1979). To support this determination, EPA published a report entitled "Radiological Impact Caused By Emissions of Radionuclides into Air in the United States Preliminary Report" (EPA 520/7-79-006, Office of Radiation Programs, U.S. EPA, Washington, D.C., August 1979).

On June 16, 1981, the Sierra Club filed suit in the U.S. District Court for the Northern District of California pursuant to the citizens' suit provision of the Act (Sierra Club v Gorsuch, No. 81-2436 WTS). The suit alleged that EPA had a nondiscretionary duty to propose standards for radionuclides under Section 112 of the Act within 180 days after listing them. On September 30, 1982, the Court ordered EPA to publish proposed regulations establishing emissions standards for radionuclides, with a notice of hearing within 180 days of the date of that order.

On April 6, 1983, EPA published a notice in the Federal Register proposing standards for radionuclide emission sources in four categories: (1) DOE facilities, (2) NRC-licensed facilities and non-DOE Federal facilities, (3) underground uranium mines, and (4) elemental phosphorus plants. Several additional categories of sources that emit radionuclides were identified, but it was determined that there were good reasons for not proposing standards for them. These source categories were (1) coal-fired boilers; (2) the phosphate industry; (3) other mineral-extraction industries; (4) uranium fuel-cycle facilities, uranium mill tailings, and high-level waste management; and (5) low-energy accelerators (48 FR

15077, April 6, 1983). To EPA's knowledge, these comprised all the source categories that release potentially regulative amounts of radionuclides to the air. To support these proposed standards and determinations, EPA published a draft report entitled "Background Information Document, Proposed Standards for Radionuclides" (EPA 520/1-83-001, Office of Radiation Programs, U.S. EPA, Washington, D.C., March 1983).

Following publication of the proposed standards, EPA held an informal public hearing in Washington, D.C., on April 28 and 29, 1983. The comment period was held open an additional 30 days to receive written comments. Subsequently, EPA received a number of requests to extend the time for submission of public comments and to conduct a public hearing on the proposed standards in the West to accommodate persons who were unable to attend the first public hearing. In response to these requests, EPA published a notice in the Federal Register that extended the comment period by an additional 45 days and held an additional informal public hearing in Denver, Colorado, on June 14, 1983 (48 FR 23665, May 26, 1983).

On February 17, 1984, the Sierra Club again filed suit in the U.S. District Court for the Northern District of California pursuant to the citizens' suit provision of the Act (Sierra Club v Ruckelshaus, No. 84-0656 WHO). The suit alleged that EPA had a nondiscretionary duty to issue final emissions standards for radionuclides or to find that they do not constitute a hazardous air pollutant (i.e., "de-list" the pollutant). In August 1984, the court granted the Sierra Club motion and ordered EPA to take final actions on radionuclides by October 23, 1984.

1.2 Purpose of the Final Background Information Document

This Background Information Document supports the Agency's final actions on radionuclides. It contains an integrated risk assessment that provides the scientific basis for these actions.

1.3 Scope of the Final Background Information Document

Volume I contains background information on radiation protection programs and a detailed description of the Agency's procedures and methods for estimating radiation dose and risk due to radionuclide emissions to the air. This material is arranged as shown in the following descriptions of the chapters:

- Chapter 2 - A summary of regulatory programs for radiation protection and the current positions of the various national and international advisory bodies and State and Federal Agencies in regard to radiation.

- Chapter 3 - A description of what makes radiation hazardous, the evidence that proves the hazard, and the evidence that relates the amount of radiation exposure to the amount of risk.

- Chapter 4 - A summary of sources that release radionuclides to the air, the physical and chemical forms of these releases, and the quantity of radionuclides that are released.
- Chapter 5 - A description of how radionuclide emissions to the air are controlled by means of emission control devices and work practices.
- Chapter 6 - A description of how radionuclides, once released into the air, move through the environment and eventually cause radiation exposure of people. This chapter also contains a description of how EPA estimates the amounts of radionuclides in the environment, i.e., in the air, on surfaces, in the food chain, and in exposed humans.
- Chapter 7 - A description of how radionuclides, once inhaled and ingested, move through the body to organs and expose these organs. This chapter also contains a description of how EPA estimates the amounts of radiation dose due to this radiation exposure of organs. It also describes how the amount of radiation dose is estimated when the source of radiation is gamma rays from a source outside of the body.
- Chapter 8 - A description of how the risk of fatal cancers and genetic effects is estimated once the amount of radiation dose is known.
- Chapter 9 - A summary of dose and risk estimates of source categories emitting significant amounts of radionuclides, which were made by using the procedures and information in the previous chapters. Associated uncertainties are discussed in the appropriate chapter, but overall uncertainties are discussed in this chapter.

Volume II contains detailed risk estimates for each source of emissions, which were performed according to the procedures given in Volume I. Each chapter contains a general description of the source category, a brief description of the processes leading to emissions of radionuclides to the air, a summary of emissions data, and estimates of radiation doses and health risks to both individuals and populations. Except for DOE facilities, each chapter also contains a brief description of emission control technology. Control technology for DOE facilities is discussed in a separate document entitled "Control Technology for Radioactive Emissions to the Atmosphere at U.S. Department of Energy Facilities" (PNL-4621, Pacific Northwest Laboratories, October 1984).

Volume II was originally issued in draft form in April 1983, when emission standards for radionuclides were proposed. In response to public comments, it has been revised and is now issued in final form.

1.4 EPA's Computer Codes

The EPA calculates doses and risks due to facilities emitting radionuclides to the air using three computer codes: AIRDOS-EPA, RADRISK, and DARTAB. These codes calculate, respectively, the resulting concentrations of radionuclides in the environment, the dose and risk to persons resulting from a given quantity of each of these radionuclides, and the total lifetime risk to individuals and the total health impact on populations. These computer codes are briefly summarized here to describe how they fit together. Details of the calculations are presented later.

The AIRDOS-EPA computer code estimates radionuclide concentrations in the air, rates of deposition on the ground, concentrations on the ground, and the amounts of radionuclides taken into the body via inhalation of air and ingestion of meat, milk, and fresh vegetables. A Gaussian plume equation predicts the atmospheric dispersion of radionuclides released from stacks or area sources. The amounts of radionuclides that are inhaled are calculated from these air concentrations and a knowledge of how much air is inhaled by an average person. The amounts of radionuclides ingested in the meat, milk, and fresh produce that people consume are estimated by coupling the output of the atmospheric transport models with the same terrestrial food chain models used by the U.S. Nuclear Regulatory Commission in Regulatory Guide 1.109. Working-level exposures are also calculated for inhalation of Rn-222 short-lived decay products.

The RADRISK code computes dose rates to organs resulting from a given quantity of a radionuclide that is ingested or inhaled. These dose rates are then used to estimate the risk of fatal cancers in an exposed cohort of 100,000 persons. All persons in the cohort are assumed to be born at the same time and to be at risk of dying from competing causes (including natural background radiation). Estimates of potential health risk due to exposure to a known quantity of approximately 500 different radionuclides are tabulated and stored until needed. These risks are summarized in terms of the probability of premature death for a member of the cohort due to a given quantity of each radionuclide that is ingested or inhaled.

The DARTAB computer code then provides estimations of the impact of radionuclide emissions from a specific facility by combining the information on the amounts of radionuclides that are ingested or inhaled (as provided by AIRDOS-EPA) with dosimetric and health effects data for a given quantity of each radionuclide (as provided by RADRISK).

The DARTAB code estimates dose and risk for individuals at user-selected locations and for population groups. Radiation doses and risks can be broken down by radionuclide, exposure pathway, and organ; or they can be summarized by direction and distance from the facility.

Chapter 2: CURRENT REGULATORY PROGRAMS AND STRATEGIES

2.1 Introduction

People have always been exposed to ionizing radiations from the cosmic rays and naturally-occurring radionuclides in the earth that make up the natural radiation background. Awareness of radiation and radioactivity dates back only to the end of the last century--to the discovery of x-rays in 1895 and the discovery of radioactivity in 1896. These discoveries mark the beginning of radiation science and the deliberate use of radiation and radionuclides in science, medicine, and industry.

The findings of radiation science rapidly led to the development of medical and industrial radiology, nuclear physics, and nuclear medicine. By the 1920's, the use of x-rays in diagnostic medicine and industrial applications was widespread, and radium was being used by industry for luminescent dials and by doctors in therapeutic procedures. By the 1930's, biomedical and genetic researchers were studying the effects of radiation on living organisms, and physicists were beginning to understand the mechanisms of spontaneous fission and radioactive decay. By the 1940's, a self-sustaining fission reaction was demonstrated, which led directly to the construction of the first nuclear reactors and atomic weapons.

Developments since the end of World War II have been rapid. Today the use of x-rays and radioactive materials is widespread and includes:

- Nuclear reactors, and their supporting fuel-cycle facilities, generate electricity; power ships and submarines; produce radioisotopes for research, space, defense, and medical applications; and are used as research tools for nuclear engineers and physicists.

- Particle accelerators produce radioisotopes and are used as research tools for studying the structure of materials and atoms.

- The radiopharmaceutical industry provides the radioisotopes needed for biomedical research and nuclear medicine.

- Nuclear medicine has developed as a recognized medical specialty in which radioisotopes are used in the diagnosis and treatment of numerous diseases.
- X-rays are widely used as a diagnostic tool in medicine and in such diverse industrial fields as oil exploration and nondestructive testing.
- Radionuclides are used in such common consumer products as luminous-dial wristwatches and smoke detectors.

The following sections of this chapter provide a brief history of the evolution of radiation protection philosophy and an outline of the current regulatory programs and strategies of the government agencies responsible for assuring that radiation and radionuclides are used safely.

2.2 The International Commission on Radiological Protection and the National Council on Radiation Protection and Measurements

Initially, the dangers and risks posed by x-rays and radioactivity were little understood. By 1896, however, "x-ray burns" were being reported in the medical literature, and by 1910, it was understood that such "burns" could also be caused by radioactive materials. By the 1920's, sufficient direct evidence (from the experiences of radium dial painters, medical radiologists, and miners) and indirect evidence (from biomedical and genetic experiments with animals) had been accumulated to persuade the scientific community that an official body should be established to make recommendations concerning human protection against exposure to x-rays and radium.

At the Second International Congress of Radiology meeting in Stockholm, Sweden, in 1928, the first radiation protection commission was created. Reflecting the uses of radiation and radioactive materials at the time, the body was named the International X-Ray and Radium Protection Commission and was charged with developing recommendations concerning protection from radiation. In 1950, to reflect better its role in a changing world, the commission was reconstituted and renamed the International Commission on Radiation Protection (ICRP).

During the Second International Congress of Radiology, the newly created commission suggested to the nations represented at the Congress that they appoint national advisory committees to represent their viewpoints before the ICRP, and to act in concert with the Commission in developing and disseminating recommendations on radiation protection. This suggestion led to the formation, in 1929, of the Advisory Committee on X-Ray and Radium Protection as the U.S. advisory group. This Advisory Committee, after a series of reorganizations and name changes, emerged in 1964 in its present form as the Congressionally chartered

National Council on Radiation Protection and Measurements (NCRP). The Congressional charter provides for the NCRP to:

- Collect, analyze, develop, and disseminate in the public interest information and recommendations about radiation protection and radiation quantities, units, and measurements.
- Develop basic concepts about radiation protection and radiation quantities, units, and measurements, and the application of these concepts.
- Provide a means by which organizations concerned with radiation protection and radiation quantities, units, and measurements may cooperate to effectively use their combined resources, and to stimulate the work of such organizations.
- Cooperate with the ICRP and other national and international organizations concerned with radiation protections and radiation quantities, units, and measurements.

Throughout their existence, the ICRP and the NCRP have worked together closely to develop radiation protection recommendations that reflect the current understanding of the dangers associated with exposure to ionizing radiation.

The first exposure limits adopted by the ICRP and the NCRP (ICRP34, ICRP38, and NCRP36) established 0.2 roentgen/day* as the "tolerance dose" for occupational exposure to x-rays and gamma radiation from radium. This limit, equivalent to approximately 25 rads/year as measured in air, was established to guard against the known effects of ionizing radiation on superficial tissue, changes in the blood, and "derangement" of internal organs, especially the reproductive organs. At the time the recommendations were made, high doses of radiation were known to cause observable effects and even to induce cancer. However, no such effects were observed at lower doses, and the epidemiological evidence at the time was inadequate to even imply the carcinogenic induction effects of moderate or low doses. Therefore, the aim of radiation protection was to guard against known effects, and the "tolerance dose" limits that were adopted were believed to represent the level of radiation that a person in normal health could tolerate without suffering observable effects. The concept of a tolerance dose and the recommended occupational exposure limit of 0.2 R/day for x- and gamma radiation remained in effect until the end of the 1940's. The recommendations of the ICRP and the NCRP made no mention of exposure of the general populace.

By the end of World War II, the widespread use of radioactive materials and scientific evidence of genetic and somatic effects at lower doses and dose rates suggested that the radiation protection recommendations of the NCRP and the ICRP would have to be revised downward.

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* The NCRP's recommendation was 0.1 roentgen/day measured in air. This limit is roughly equivalent to the ICRP limit, which was conventionally measured at the point of exposure and included back-scatter.

By 1948, the NCRP had formulated its position on appropriate new limits. These limits were largely accepted by the ICRP in its recommendations of 1950 and formally issued by the NCRP in 1954 (ICRP51, NCRP54). Whereas the immediate effect was to lower the basic whole-body occupational dose limit to 0.3 rad/week (approximately 15 rads/year), the revised recommendations also embodied several new and important concepts in the formulation of radiation protection criteria.

First, the recommendations recognized the differences in the effects of various types and energies of radiation; both ICRP's and NCRP's recommendations included discussions of the weighting factors that should be applied to radiations of differing types and energies. The NCRP advocated the use of the "rem" to express the equivalence in biological effect between radiations of differing types and energy.* Although the ICRP noted the shift toward the acceptance of the rem, it continued to express its recommendations in terms of the rad, with the caveat that neutrons should carry a quality factor of ten.

Second, the recommendations of both organizations introduced the concept of critical organs and tissues. The intent of this concept was to assure that no tissue or organ, with the exception of the skin, would receive a dose in excess of that allowed for the whole body. At the time, scientific evidence was lacking on which to base different recommended limits for the various tissues and organs. Thus, all blood-forming organs were considered critical organs and were limited to the same exposure as the whole body. The skin was allowed an exposure of 30 rad/year and the extremities were allowed a limit of 75 rads/year.

Third, the recommendations of the NCRP included the suggestion that individuals under the age of 18 receive no more than one-tenth the exposure allowed for adults. The reasoning behind this particular recommendation is interesting as it reflects clearly the limited knowledge of the times. The scientific evidence indicated a clear relationship between accumulated dose and genetic effect. However, this evidence was obtained exclusively from animal studies that had been conducted with doses ranging from 25 to thousands of rads. There was no evidence from exposures less than 25 rads accumulated dose, and the interpretation of the animal data and the implications for humans was unclear and did not support a specific permissible dose. The data did suggest that genetic damage was more dependent on accumulated dose than previously believed, but experience showed that exposure for prolonged periods to the permissible dose (1.0 R/week) did not result in any observable genetic effects. The NCRP decided that it was not necessary to change the occupational limit to provide additional protection beyond that provided by the reduction in the permissible dose limit to 0.3 R/week. At the same time, it recommended limiting the exposure of individuals under the age of 18 to assure that they did not accumulate a genetic dose that would later preclude their employment as radiation workers. The factor of ten was rather arbitrary, but was believed to be sufficient to protect the future employability of all individuals (NCRP54).

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* The exact relationship between roentgens, rads, and rems is beyond the scope of this work. In simple terms, the roentgen is a measure of the degree of ionization induced by x- and gamma radiations in air. The rad (radiation absorbed dose) is a measure of the energy imparted to matter by radiation. And the rem (roentgen equivalent man) is a measure of equivalence for the relative biological effect of radiations of different types and energies on man. Over the range of energies typically encountered, the relationship of roentgens to rads to rems for x- and gamma radiation is essentially equality. For beta radiation, rads are equivalent to rems. And for alpha radiation one rad equals 10 to 20 rems.

Fourth, the concept of a tolerance dose was replaced by the concept of a maximum permissible dose. The change in terminology reflected the increasing awareness that any radiation exposure might involve some risk and that repair mechanisms might be less effective than previously believed. Therefore, the concept of a maximum permissible dose (expressed as dose per unit of time) was adopted because it better reflects the uncertainty in our knowledge than does the concept of tolerance dose. The maximum permissible dose was defined as the level of exposure that entailed a small risk compared with those posed by other hazards in life (ICRP51).

Finally, in explicit recognition of the inadequacy of our knowledge regarding the effects of radiation and of the possibility that any exposure might have some potential for harm, the recommendations included an admonition that every effort should be made to reduce exposure to all kinds of ionizing radiation to the lowest possible level. This concept, known originally as ALAP (as low as practicable) and later as ALARA (as low as reasonably achievable), would become a cornerstone of radiation protection philosophy.

During the 1950's, a great deal of scientific evidence on the effects of radiation became available from studies of the radium dial painters, radiologists, and the survivors of the atomic bombs dropped on Japan. This evidence suggested that genetic effects and long-term somatic effects were more important than previously considered. Thus, by the late 1950's, the ICRP and NCRP recommendations were again revised (ICRP59, NCRP59). These revisions include the following major changes: the maximum permissible dose for whole-body exposure and the most critical organs (blood-forming organs, gonads, and the lens of the eye) was lowered to 5 rems/year, with a quarterly limit of 3 rems; the limit for exposure of other organs was set at 30 rems/year; internal exposures were controlled by a comprehensive set of maximum permissible concentrations of radionuclides in air and water based on the most restrictive case of a young worker; and recommendations were included for some nonoccupational groups and for the general population (for the first time).

The lowering of the maximum permissible whole-body dose from 0.3 rad/week to 5 rems/year, with a quarterly limit of 3 rems, reflects both the new evidence and the uncertainties of the time. Although no adverse effects were observed among workers who had received the maximum permissible dose of 0.3 rad/week, there was concern that the lifetime accumulation of as much as 750 rads (15 rads/year times 50 years) was too much. Lowering the maximum permissible dose by a factor of three was believed to provide a greater margin of safety. At the same time, operational experience showed that 5 rems/year could be met in most instances, particularly with the additional operational flexibility provided by expressing the limit on an annual and quarterly basis.

The recommendations given for nonoccupational exposures were based on concerns of genetic effects. The evidence available suggested that genetic effects were primarily dependent on the total accumulated dose. Thus, having sought the opinions of respected geneticists, the ICRP and the NCRP adopted the recommendation that accumulated gonadal dose to age 30 be limited to 5 rems from sources other than natural background and medical exposure. As an operational guide, the NCRP recommended that the maximum dose to any individual be limited to 0.5 rem/year, with maximum permissible body burdens of radionuclides (to control internal exposures) set at one-tenth that allowed for radiation workers. These values were derived from consideration of the genetically significant dose to the population, and were established "primarily for the purpose of keeping the average dose to the whole population as low as reasonably possible, and not because of the likelihood of specific injury to the individual" (NCRP59).

During the 1960's, the ICRP and NCRP again lowered the maximum permissible dose limits (ICRP65, NCRP71). The considerable scientific data on the effects of exposure to ionizing radiation were still inconclusive with respect to the dose-response relationship at low exposure levels; thus, both organizations continued to stress the need to keep all exposures to the lowest possible level.

The NCRP and the ICRP made the following similar recommendations:

- Limit the dose to the whole-body, red bone marrow, and gonads to 5 rems in any year, with a retrospective limit of 10 to 15 rems in any given year as long as total accumulated dose did not exceed 5X(N-18), where N is age in years.
- Limit the dose to the skin, hands, and forearms to 15, 75, and 30 rems per year, respectively.
- Limit the dose to any other organ or tissue to 15 rems per year.
- Limit the dose to any non-occupationally exposed individual in the population to 0.5 rem per year.
- Limit the average dose to the population to 0.17 rem per year.

The scientific evidence and the protection philosophy on which the above recommendations were based were set forth in detail in NCRP71. In the case of occupational exposure limits, the goal of protection was to ensure that the risks of genetic and somatic effects were small enough to be comparable to the risks experienced by workers in other safe industries. The conservatively derived numerical limits recommended were based on the linear, no-threshold, dose-response model, and were believed to represent a level of risk that was readily acceptable to an average individual. For nonoccupational exposures, the goal of protection was to ensure that the risks of genetic or somatic effects were small compared with other risks encountered in everyday life. The derivation of specific limits was complicated by the unknown dose-response relationship at low exposure levels and the fact that the risks of radiation exposure did not necessarily accrue to the same individuals who benefited from the activity responsible for the exposure. Therefore, it was necessary to derive limits that gave adequate protection to each member of the public and to the gene pool of the population as a whole, while still allowing the development of beneficial uses of radiation and radionuclides.

In 1977, the ICRP made a fundamental change in its recommendations when it abandoned the critical organ concept in favor of the weighted whole-body dose equivalent concept for limiting occupational exposure (ICRP77). The change, made to reflect our increased understanding of the differing radiosensitivity of the various organs and tissues, did not affect the overall limit of 5 rems per year, and is not intended to be applied to nonoccupational exposures.

Also significant is the fact that ICRP's 1977 recommendations represent the first explicit attempt to relate and justify permissible radiation exposures with quantitative levels of acceptable risk. Thus, average occupational exposures (approximately 0.5 rem/year) are equated with risks in safe industries, given as 10^{-4} annually. At the maximum limit of 5 rems/year, the risk is equated with that experienced by some workers in recognized hazardous occupations. Similarly, the risks implied by the nonoccupational limit of 0.5 rem/year are equated to levels of risk of less than 10^{-2} in a lifetime; the general populace's average exposure is equivalent to a lifetime risk on the order of 10^{-3} to 10^{-4}. The ICRP believed these levels of risk were in the range that most individuals find acceptable.

The NCRP has not formally changed its recommendations for occupational exposure to correspond to the 1977 recommendations of the ICRP. It has been working diligently, however, to review its recommendations, and has circulated a draft of proposed changes to various interested scientists and regulatory bodies for their comment. The relevant nonoccupational exposure limits are:

- 0.5 rem/year whole-body dose equivalent, not including background or medical radiation, for individuals in the population when the exposure is not continuous.

- 0.1 rem/year whole-body dose equivalent, not including background or medical radiation, for individuals in the population when the exposure is continuous.

- Continued use of a total dose limitation system based on justification of every exposure and application of the "as low as reasonably achievable" philosophy to every exposure.

The NCRP equates continuous exposure at the level of 0.1 rem/year to a lifetime risk of developing cancer of about one in a thousand. The NCRP has not formulated exposure limits for specific organs, but it notes that the permissible limits will necessarily be higher than the whole-body limit in inverse ratio of the risk for a particular organ to the total risk for whole-body exposure. In response to EPA's proposed national emission standards for radionuclides, the NCRP suggested that since the 0.1 rem/year limit is the limit for all exposures from all sources (excluding natural background and medical radiation), the operator of any site responsible for more than 25 percent of the annual limit be required to assure that the exposure of the maximally exposed individual is less than 0.1 rem/year from all sources (NCRP 84).

2.3 Federal Guidance

The ICRP and the NCRP function as nongovernmental advisory bodies. Their recommendations are not binding on any user of radiation or radioactive materials. The wealth of new scientific information on the effects of radiation that became available in the 1950's prompted the President to establish an official government entity with responsibility for formulating radiation protection criteria and coordinating radiation protection activities. Thus, the Federal Radiation Council was established in 1959 by Executive Order 10831. The Council included representatives from all of the Federal agencies concerned with radiation protection, and acted as a coordinating body for all of the radiation activities conducted by the Federal government. In addition to its coordinating function, the Council's major responsibility was to "...advise the President with respect to radiation matters, directly or indirectly affecting health, including guidance for all Federal agencies in the formulation of radiation standards and in the establishment and execution of programs of cooperation with States..." (FRC60).

The Council's first recommendations concerning radiation protection standards for Federal agencies were approved by the President in 1960. Based largely on the work and recommendations of the ICRP and the NCRP, the guidance established the following limits for occupational exposures:

- Whole body, head and trunk, active blood forming organs, gonads, or lens of eye--not to exceed 3 rems in 13 weeks and total accumulated dose limited to 5 times the number of years beyond age 18.

- Skin of whole body and thyroid--not to exceed 10 rems in 13 weeks or 30 rems per year.

- Hands, forearms, feet, and ankles--not to exceed 25 rems in 13 weeks or 75 rems per year.
- Bone--not to exceed 0.1 microgram of Radium-226 or its biological equivalent.
- Any other organ--not to exceed 5 rems per 13 weeks or 15 rems per year.

Although these levels differ slightly from those recommended by NCRP and ICRP at the time, the differences do not represent any greater or lesser protection. In fact, the FRC not only accepted the levels recommended by the NCRP for occupational exposure, it adopted the NCRP's philosophy of acceptable risk for determining occupational exposure limits. Although quantitative measures of risk were not given in the Guidance, the prescribed levels were not expected to cause appreciable bodily injury to an individual during his or her lifetime. Thus, while the possibility of some injury was not zero, it was so low as to be acceptable if there was any significant benefit derived from the exposure.

The guidance also established exposure limits for members of the public. These were set at 0.5 rem per year (whole body) for an individual, and an average of 5 rems in 30 years (gonadal) per capita. The guidance also provided for developing a suitable sample of the population as an operational basis for determining compliance with the limit when doses to all individuals are unknown. Exposure to this population sample was not to exceed 0.17 rem per capita per year. The population limit of 0.5 rem to any individual per year, was derived from consideration of natural background exposure. Natural background radiation varies by a factor of two to four from location to location.

In addition to the formal exposure limits, the guidance also established as Federal policy that there should be no radiation exposure without an expectation of benefit, and that "every effort should be made to encourage the maintenance of radiation doses as far below this guide as practicable." The inclusion of the requirements to consider benefits and keep all exposure to a minimum was based on the possibility that there is no threshold dose for radiation. The linear non-threshold dose response was assumed to place an upper limit on the estimate of radiation risk. However, the FRC explicitly recognized that it might also represent the true level of risk. If so, then any radiation exposure carried some risk, and it was necessary to avoid all unproductive exposures and to keep all productive exposures as "far below this guide as practicable."

In 1967, the Federal Radiation Council issued guidance for the control of radiation hazards in uranium mining (FRC67). The need for such guidance was clearly indicated by the epidemiological evidence that showed a higher incidence of lung cancer in adult males who worked in uranium mines compared with the incidence in adult males from the same locations who had not worked in mines. The Guidance established specific exposure limits and recommended that all exposures be kept as far below the guide limits as possible. The limits chosen represented a trade-off between the risks incurred at various exposure levels, the technical feasibility of reducing the exposure, and the benefits of the activity responsible for the exposure. The guidance also applied to nonuranium mines.

In 1970, the functions of the Federal Radiation Council were transferred to the U.S. Environmental Protection Agency. In 1971, the EPA revised the Federal Guidance for the control of radiation hazards in uranium mining (EPA71). Based on the risk levels associated with the exposure limits established in 1967, the upper limit of exposure was reduced by a factor of three. The EPA has also provided Federal Guidance for the Diagnostic Use of X-Rays (EPA78). This guidance establishes maximum skin entrance doses for various types of routine x-ray examinations. It also establishes the requirement that all x-ray exposures be based on clinical indication and diagnostic need, and that all exposure of patients should be kept as low as reasonably achievable consistent with the diagnostic need.

In 1981, the EPA proposed new Federal Guidance for Occupational Exposures to supersede the 1960 guidance (EPA81). The 1981 recommended guidance follows the principles set forth by the ICRP in 1977, with respect to combining internal and external doses. The basic occupational limit suggested in the guidance is 5 rems per year. This recommended guidance has not been adopted as Federal policy. The proposals in the guidance were issued for public comment in 1981 and are currently being reviewed and revised in light of the comments received.

2.4 The Environmental Protection Agency

In addition to the statutory responsibility to provide Federal guidance on radiation protection, the Environmental Protection Agency (EPA) has various statutory authorities and responsibilities regarding regulation of exposure to radiation. The standards and the regulations that EPA has promulgated and proposed with respect to controlling radiation exposures are summarized here.

The U.S. Atomic Energy Act of 1954, as amended, and Reorganization Plan No. 3 granted EPA the authority to establish generally applicable environmental standards for exposure to radionuclides. Pursuant to this authority, in 1977 the EPA issued standards limiting exposure from operations of the light-water reactor nuclear fuel cycle (EPA77). These standards cover normal operations of the uranium fuel cycle, excluding mining and spent-fuel disposal. The standards limit the annual dose equivalent to any member of the public from all phases of the uranium fuel cycle (excluding radon and its daughters) to 25 mrems to the whole body, 75 mrems to the thyroid, and 25 mrems to any other organ. To protect against the buildup of long-lived radionuclides in the environment, the standard also sets normalized emission limits for krypton-85, iodine-129, and plutonium-239 combined with other transuranics with a half-life exceeding one year. The dose limits imposed by the standard cover all exposures resulting from releases to air and water from operations of fuel-cycle facilities.

The development of this standard took into account both the maximum risk to an individual and the overall effect of releases from fuel-cycle operations on the population and balanced these risks against the costs of effluent control in a primarily qualitative way.

Under the authority of the Uranium Mill Tailings Radiation Control Act, the EPA has promulgated standards limiting public exposure to radiation from uranium tailings piles (EPA83a, EPA83b). Whereas the standards for inactive and active tailings piles differ, a consistent basis is used for these standards. Again, the Agency sought to balance the radiation risks imposed on individuals and the population in the vicinity of the pile against the feasibility and costs of control.

Under the authority of the U.S. Atomic Energy Act of 1954, as amended, the EPA has proposed standards for disposal of spent fuel, high-level wastes, and transuranic elements (EPA82). The proposed standard establishes two different limits: (1) during the active waste-disposal phase, operations must be conducted so that no member of the public receives a dose greater than that allowed for other phases of the uranium fuel cycle; and (2) once the repository is closed, exposure is to be controlled by limiting releases. The release limits were derived by summing, over long time periods, the estimated risks to all persons exposed to radioactive materials released into the environment. The uncertainties involved in estimating the performance of a theoretical repository led to this unusual approach, and the proposed standard admonishes the agencies responsible for constructing and operating such repositories to take steps to reduce releases below the upper bounds given in the standard to the extent reasonably achievable.

Under the authority of the Safe Drinking Water Act, the EPA has issued interim regulations covering the permissible levels of radium, gross alpha and manmade beta, and photon emitting contaminants in community water systems (EPA76). The limits are expressed in picocuries/liter. The limits chosen for manmade beta and photon emitters equate to approximately 4 mrems/year whole-body or organ dose to the most exposed individual. In the background information for the standard, the 4 mrems/year exposure through a single pathway that the standard permits is explicitly compared with the overall population standard of 170 mrems/year, and the conclusion is expressed that the roughly 40-fold decrease is appropriate for a single pathway.

Section 122 of the Clean Air Act amendments of 1977 (Public Law 95-95) directed the Administrator of EPA to review all relevant information and determine if emissions of hazardous pollutants into air will cause or contribute to air pollution that may reasonably be expected to endanger public health. In December 1979, EPA designated radionuclides as hazardous air pollutants under Section 112 of the Act. On April 6, 1983, EPA published proposed National Emission Standards for radionuclides for selected sources in the Federal Register (48 CFR 15076).

2.5 Nuclear Regulatory Commission

Under the authority of the Atomic Energy Act of 1954, as amended, the Nuclear Regulatory Commission (NRC) is responsible for licensing and regulating the use of byproduct, source, and special nuclear material, and for assuring that all licensed activities are conducted in a manner that protects public health and safety. The Federal guidance on radiation protection applies directly to the NRC; therefore, the NRC must assure that none of the operations of its licensees exposes an individual of the public to more than 0.5 rem/year. The dose limits imposed by the EPA's standard for uranium fuel-cycle facilities also apply to the fuel-cycle facilities licensed by the NRC. These facilities are prohibited from releasing radioactive effluents in amounts that would result in doses greater than the 25 mrems/year limit imposed by that standard.

The NRC exercises its statutory authority by imposing a combination of design criteria, operating parameters, and license conditions at the time of construction and licensing. It assures that the license conditions are fulfilled through inspection and enforcement. The NRC licenses more than 7000 users of radioactivity. The regulation of fuel-cycle licensees is discussed separately from the regulation of byproduct material licensees.

2.5.1 Fuel Cycle Licenses

The NRC does not use the term "fuel cycle facilities" to define its classes of licensees. The term is used here to coincide with the EPA use of the term in its standard for uranium fuel cycle facilities. As a practical matter, this term includes the NRC's large source and special nuclear material, and production and utilization facilities. The NRC's regulations require an analysis of probable radioactive effluents and their effects on the population near fuel cycle facilities. The NRC also assures that all exposures are as low as reasonably achievable by imposing design criteria and specific equipment requirements on the licensees. After a license has been issued, fuel-cycle licensees must monitor their emissions and take environmental measurements to assure that the design criteria and license conditions have been met. For practical purposes, the NRC adopted the maximum permissible concentrations developed by the NCRP to relate effluent concentrations to exposure.

In the 1970's, the NRC formalized the implementation of as low as reasonably achievable exposure levels by issuing a regulatory guide for as low as reasonably achievable design criteria. This coincided with a decision to adopt, as a design criterion, a maximum permissible dose of 5 mrems/year from a single nuclear electric generating station. The 5-mrem limit applies to the most exposed individual actually living in the vicinity of the reactor, and refers to whole-body doses from external radiation by the air pathway (NRC77).

2.5.2 Byproduct Material Licenses

The NRC's licensing and inspection procedure for byproduct material users is less uniform than that imposed on major fuel-cycle licensees for two reasons: (1) the much larger number of such licensees, and (2) the much smaller potential for releasing significant quantities of radioactive materials into the environment. The prelicensing assurance procedures of imposing design reviews, operating practices, and license conditions prior to construction and operation are similar. The amount of protection that is afforded the public from releases of radioactive materials from these facilities can vary considerably because of three factors. First, the requirements that the NRC imposes for monitoring effluents and environmental radioactivity are much less stringent for these licensees. If the quantity of materials handled is small enough, the NRC might not impose any monitoring requirements. Second, and more important, the level of protection can vary considerably because where the licensee must meet the effluent concentrations for an area of unrestricted access is not consistently defined. Depending on the particular licensee, this area has been defined as the nearest inhabited structure, as the boundary of the user's property line, as the roof of the building where the effluents are vented, or as the mouth of the stack or vent. Finally, not all users are allowed to reach 100 percent of the permissible concentrations in their effluents. In fact, the NRC has implemented as low as reasonably achievable considerations on many of these licensees by limiting them to 10 percent of the maximum permissible concentration in their effluents.

2.6 Department of Energy

The U.S. Department of Energy (DOE) operates a complex of national laboratories and weapons facilities. These facilities are not licensed by the NRC. The DOE is responsible, under the U.S. Atomic Energy Act of 1954, as amended, for assuring that these facilities are operated in a manner that does not jeopardize public health and safety.

For practical purposes, the DOE has adopted the NCRP's maximum permissible concentrations in air and water as a workable way to assure that the dose limits of 0.5 rem/year whole-body and 1.5 rems/year to any organ are being observed. The DOE also has a requirement that all doses be kept as low as is reasonably achievable, but the contractors that operate the various DOE sites have a great deal of latitude in implementing policies and procedures to assure that all doses are kept to the lowest possible level.

The DOE assures that its operations are within its operating guidelines by requiring its contractors to maintain radiation monitoring systems around each of its sites and to report the results in an annual summary report. New facilities and modifications to existing facilities are subject to extensive design criteria reviews (similar to those used by the NRC). During the mid-1970's, the DOE initiated a systematic effluent-reduction program that resulted in the upgrading of many facilities and effected a corresponding reduction in the effluents (including airborne and liquid radioactive materials) released to the environment.

2.7 Other Federal Agencies

The Department of Defense operates several nuclear installations, including a fleet of nuclear-powered submarines and their shore support facilities. The DOD, like other Federal agencies, must comply with the Federal radiation guidance. The DOD has not formally adopted any more stringent exposure limits for members of the public than the 0.5 rem/year allowed by the Federal guidance.

2.7.1 Center for Medical Devices and Radiological Health

Under the Radiation Control Act of 1968, the major responsibility of the Center for Medical Devices and Radiological Health in the area of radiation protection is the specification of performance criteria for electronic products, including x-ray equipment and other medical devices. This group also performs environmental sampling in support of other agencies, but no regulatory authority is involved.

2.7.2 Mine Safety and Health Administration

The Mine Safety and Health Administration (MSHA) has the regulatory authority to set standards for exposures of miners to radon and its decay products and other (nonradiological) pollutants in mines. The MSHA has adopted the Federal guidance for exposure of uranium miners (EPA71). It has no authority or responsibility for protecting members of the general public from the hazards associated with radiation.

2.7.3 Occupational Safety and Health Administration

The Occupational Safety and Health Administration (OSHA) is responsible for assuring a safe work place for all workers. This authority, however, does not apply to radiation workers at government-owned or NRC-licensed facilities. This group does have the authority to set exposure limits for workers at unlicensed facilities, such as particle accelerators, but it does not have any authority to regulate public exposure to radiation. OSHA has adopted the occupational exposure limits of the NRC, except it has not imposed the requirement to keep all doses as low as is reasonably achievable.

2.7.4 Department of Transportation

The Department of Transportation (DOT) has statutory responsibility for regulating the shipment and transportation of radioactive materials. This authority includes the responsibility to protect the public from exposure to radioactive materials while they are in transit. For practical purposes, the DOT has implemented its authority through the specification of performance standards for shipment containers, and by setting maximum exposure rates at the surface of any package containing radioactive materials. These limits were set to assure compliance with the Federal guidance for occupational exposure, and they are believed to be sufficient to protect the public from exposure. The DOT also controls potential public exposure by managing the routing of radioactive shipments to avoid densely populated areas.

2.8 State Agencies

States have important authority for protecting the public from the hazards associated with ionizing radiation. In 26 states, the states have assumed NRC's inspection, enforcement, and licensing responsibilities for users of source and byproduct materials and users of small quantities of special nuclear material. These "NRC-agreement states," which license and regulate more than 11,500 users of radiation and radioactive materials, are bound by formal agreements to adopt requirements consistent with those imposed by the NRC. The NRC continues to perform this function for all licensable uses of source, byproduct, and special nuclear material in the 24 states that are not agreement states.

Nonagreement states, as well as NRC-agreement states, regulate the exposures to workers from electronic sources of radiation. Also, all states retain the authority to regulate the use of naturally occurring (i.e., radium) and accelerator-produced radioactive materials.

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Chapter 3: HAZARD IDENTIFICATION

The adverse biological reactions associated with ionizing radiations, and hence with radioactive materials, are carcinogenicity, mutagenicity, and teratogenicity. Carcinogenicity is the ability to produce cancer. Mutagenicity is the property of being able to induce genetic mutation, which may be in the nucleus of either somatic (body) or germ (reproductive) cells. Teratogenicity refers to the ability of an agent to induce or increase the incidence of congenital malformations as a result of permanent structural or functional deviations produced during the growth and development of an embryo (these are more commonly referred to as birth defects).

Ionizing radiation causes injury by breaking constituent body molecules into electrically charged fragments called "ions" and thereby producing chemical rearrangements that may lead to permanent cellular damage. The degree of biological damage caused by various types of radiation varies according to how close together the ionizations occur. Some ionizing radiations (e.g., alpha particles) produce intense regions of ionization. For this reason they are called high-LET (linear energy transfer) particles. Other types of radiation [such as high-energy photons (x-rays)] that release electrons that cause ionization and beta particles are called low-LET radiations because of the sparse pattern of ionization they produce. In equal doses, the carcinogenicity and mutagenicity of high-LET radiations are generally an order of magnitude or more greater than for low-LET radiations.

Radium, radon, radon daughters, and several other naturally occurring radioactive materials emit alpha particles; thus, when these materials are ingested or inhaled, they are a source of high-LET particles within the body. Man-made radionuclides are usually beta and photon emitters of low-LET radiations. Notable exceptions to this generalization are plutonium and other transuranium radionuclides, most of which emit alpha radiation.

3.1 Evidence That Radiation Is Carcinogenic

The production and properties of x-rays were demonstrated within one month of the public reporting of Roentgen's discovery of x-rays. The first report of acute skin injury was made in 1896 (Mo67). The first human cancer attributed to this radiation was reported in 1902 (Vo02). By 1911, 94 cases of radiation-related skin cancer and 5 cases of leukemia in man had been reported in the literature (Up75). Efforts to study this phenomenon through the use of experimental animals produced the first reported radiation-related cancers in experimental animals in 1910 and 1912 (Maa10, Maa12). Since that time, an extensive body of literature has evolved on radiation carcinogenesis in man and animals. This literature has been reviewed most recently by the United Nations Scientific Committee on the Effects of Atomic Radiation (UNSCEAR), and by the National Academy of Sciences Advisory Committee on the Biological Effects of Ionizing Radiations (NAS-BEIR Committee) (UNSCEAR82, NAS80).

Identification of the carcinogenicity of radioactive emissions followed a parallel course. The first association of inhaled radioactive material and carcinogenesis in man was made by Uhlig in 1921 in a study of radon exposure and lung cancer in underground miners in the Erz Mountains (Uh21). This association was reaffirmed by Ludewig and Lorenser in 1924 (Lu24)). Ingestion of radioactive materials was also demonstrated to be a pathway for carcinogenesis in man. As early as 1925 ingested radium was known to cause bone necrosis (Ho25), and in 1929 the first report was published on the association of radium ingestion and osteogenic sarcoma (Mab29).

The expected levels of exposure to radioactive pollutants in the environment are too low to produce an acute (immediate) response. Their effect is more likely to be a delayed response, in the form of an increased incidence of cancer long after exposure. An increase in cancer incidence or mortality with increasing radiation dose has been demonstrated for many types of cancer in both human populations and laboratory animals (UNSCEAR77,82). Studies of humans exposed to internal or external sources of ionizing radiation have shown that the incidence of cancer increases with increased radiation exposure. This increased cancer, however, is usually associated with appreciably greater doses and exposure frequencies than those encountered in the environment. Malignant tumors most often appeared long after the radiation exposure, usually 10 to 35 years later (NAS80, UNSCEAR82). The tumors appeared in various organs. In the case of internal sources of radiation due to radioactive materials, the metabolism of the materials generally leads to their deposition in specific organs and results in a higher-than-normal risk of cancer in these organs.

Whereas many, if not most, chemical carcinogens appear to be organ- or tissue-specific, ionizing radiation can be considered pancarcinogenic. According to Storer (Stb75): "Ionizing radiation in sufficiently high dosage acts as a complete carcinogen in that it serves as both initiator and promoter. Further, cancers can be induced in nearly any tissue or organ of man or experimental animals by the proper choice of radiation dose and exposure schedule." Radiation-induced cancers in humans have been reported in the following tissues: thyroid, female breast, lung, bone marrow (leukemia), stomach, liver, large intestine, brain, salivary glands, bone, esophagus, small intestine, urinary bladder, pancreas, rectum, lymphatic tissues, skin, pharynx, uterus, ovary, mucosa of cranial sinuses, and kidney (UNSCEAR77,82; NAS72,80; Be77, Ka82, Wa83).

A number of studies of populations exposed to high levels of radiation have identified which organs are at greatest risk following radiation exposure. Brief discussions of these findings follow.

1. Atomic Bomb Survivors - The survivors of the atomic bomb explosions at Hiroshima and Nagasaki, Japan, were exposed to whole-body external radiation doses of 0 to more than 200 rads.* An international group has been observing the population since 1950. The most recent reports published by this group (Ka82, Wa83) indicate that an increase in cancer mortality has been shown for many cancers, leukemia, thyroid, breast, lung cancer, esophogeal and stomach cancer, colon cancer, cancer of urinary organs, and multiple myeloma.

2. Ankylosing Spondylitics - A large group of patients were given x-ray therapy for ankylosing spondylitis of the spine during the years 1934 to 1954. X-ray doses usually exceeded 100 rad. British investigators have been following this group since about 1957. The most recent review of the data shows excess cancers in irradiated organs, including leukemia, lymphoma, lung and bone cancer, and cancer of the pharynx, esophagus, stomach, pancreas, and large intestine (UNSCEAR77, NAS80).

3. Mammary Exposure - Several groups of women who were exposed to x-rays during diagnostic radiation of the thorax or during radiotherapy for conditions involving the breast have been studied. Although most of the groups have been followed only a relatively short time (about 15 years), a significant increase in the incidence of breast cancer has been observed (UNSCEAR77). The dose that produced these effects averaged about 100 rads.

4. Medical Treatment of Benign Conditions - Several groups of persons who were medically treated with x-rays to alleviate some benign conditions have been studied. Excess cancer has developed in many of the organs irradiated (e.g., breast, brain, thyroid, and probably salivary glands, skin, bone, and pelvic organs) following doses ranging from less than 10 to more than 100 rads (UNSCEAR77). Excess leukemia has also occurred in some groups. The followup period for most groups has been short, often less than 20 years.

5. Underground Miners - Studies of excess cancer mortality in U.S. underground miners exposed to elevated levels of radon started in the 1950's and 1960's. Groups that have worked in various types of mines, including uranium and fluospar, are being studied in the United States, Canada, Great Britain, Sweden, China, and Czechoslovakia. Most of the miners studied have been subjected to high rates of exposure; however, a recent review indicates increased incidence of lung cancer has been observed in some miners exposed

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* The rad is the unit of dose in common use; 1 rad equals 100 ergs of absorbed energy per gram of material.

at cumulative levels approximating those that can occur wherever high environmental concentrations of radon are present (NAS80). The dose response shown in all the study groups is nearly proportional to the dose (NAS80).

6. Ingested or Injected Radium - Workers who ingested Ra-226 while painting clock dials have been studied for 35 to 45 years, and patients who received injections of Ra-226 or Ra-224 for medical purposes have been studied for 20 to 30 years (NAS72,80). Excess incidence of leukemia and osteosarcoma related to Ra-224 exposure has been observed. Calculated cumulative average doses for these study groups ranged from 200 to 1700 rads. A study now under way that deals with exposure levels under 90 rads should provide additional data (NAS80).

7. Injected Thorotrast - Medical use of Thorotrast (colloidal thorium dioxide) as an x-ray contrast medium introduced radioactive thorium and its daughters into a number of patients. Research studies have followed patients in Denmark, Portugal, Japan, and Germany for about 40 years and patients in the United States for about 10 years (UNSCEAR77, NAS80). An increased incidence of liver, bone, and lung cancer has been reported in addition to increased anemia, leukemia, and multiple myeloma (In79). Calculated cumulative doses range from tens to hundreds of rads.

8. Diagnostic X-ray Exposure During Pregnancy - Effects of x-ray exposure of the fetus during pregnancy have been studied in Great Britain since 1954, and several retrospective studies have been made in the United States since that time (NAS80, UNSCEAR77). Increased incidence of leukemia and other childhood cancers may be induced in populations exposed to absorbed doses of 0.2 to 20 rads _in utero_ (NAS80, UNSCEAR77).

Not all of the cancers induced by radiation are fatal. The fraction of fatal cancers is different for each type of cancer. The BEIR-3 committee estimated the fraction of fatal cancers by site and sex (NAS80). Estimates of cancers by site ranged from about 20 percent fatal in the case of thyroid cancer to 100 percent fatal in the case of liver cancer. They concluded that, on the average, females have 2.00 times as many total cancers as fatal cancers following radiation exposure, and males have 1.5 times as many (NAS80). Although many of the radiation-induced cancers are not fatal, they still are costly and adversely affect the persons life style for the remainder of his or her life span. Just how these costs and years of impaired life should be weighed in evaluating the hazards of radiation exposure is not certain. In this assessment, only the risk of fatal carcinogenesis is addressed.

In addition to the evidence that radiation is a pancarcinogen and as such can induce cancers in nearly any tissue or organ, it can also induce cancer by any route of exposure (dermal, inhalation, ingestion, and injection).

Inhalation is likely to be the major route of environmental exposure to airborne radioactive pollutants, and the principal organ at risk is likely to be the lung. Some radiation exposure to airborne pollutants by the ingestion route is possible, however, as these pollutants are deposited on soil, on plants, or in sources of water. Ingestion of inhaled particulate also occurs. Some radionuclides may also cause whole-body gamma radiation exposure while airborne or after deposition on the ground.

Estimates of cancer risk are based on the absorbed dose of radiation in an organ or tissue. Given the same type of radiation, the risk for a particular dosage would be the same, regardless of the source of the radiation. Numerical estimates of the cancer risk posed by a unit dose of radiation in various organs and tissues are presented in Chapter 8. The models used to calculate radiation doses from a specific source are described in Chapters 6 and 7.

The overwhelming body of epidemiological (human) data makes it unnecessary to base major conclusions concerning the risk of radiation-induced cancers on evidence provided by animal tests; however, these data are relevant to the interpretation of human data (NAS80) and contribute additional evidence to the epidemiological data base for humans. Radiation-induced cancers have been demonstrated in several animal species, including rats, mice, hamsters, guinea pigs, cats, dogs, sheep, cattle, pigs, and monkeys. Induced through multiple routes of administration and at multiple dose levels, these cancers have occurred in several organs or tissues. These animal studies have provided information on the significance of dose rate compared with the age of the animals at exposure, the sex of the animals, and the genetic characteristics of the test strain. They have shown that radiation-induced cancers become detectable after varying latent periods, sometimes several years after exposure. The studies further show that the total number of cancers that eventually develop varies consistently with the size of the dose each animal receives. Experimental studies in animals have also established that the carcinogenic effect of high-LET radiation (alpha radiations or neutrons) is greater than that of low-LET radiation (x-rays or gamma rays).

A number of researchers have induced transformations in mammalian tissue culture, including the embryo cells of mice and hamsters (Bo84, Ke84, Ha84, Gu84). Researchers have found that the DNA molecule is the carrier of radiation-induced transformations and that the radiation causes alterations in specific segments of genetic information (Bo84). Kennedy and Little have postulated that radiation-induced cell transformation is a two-step process (Ke84). In the first step, an alteration frequently occurs in a large fraction of the cells exposed to a large dose (600-rad) or to a low dose (100-rad) and a promoting agent. The second step is a rare event that occurs in one cell out of the million cells that are produced from the irradiated cells and involves the malignant transformation of that cell. This transformation occurs randomly during the growth stage of irradiated cultures. A significant finding of this research is that the process involved in the malignant transformation of mouse embryo cells caused by radiation is similar to that caused by chemical carcinogens. Another major finding of recent research (Gu84) is that DNA from radiation-induced mouse tumors contains an activated oncogene that can transform specific types of cultured cells when introduced into these cells. The researchers also found that a difference in only one base in the oncogene was responsible for the transformation. Thus, radiation can induce tumors even when only a small change in the DNA occurs as a result of irradiation.

In like concentrations, radioactive materials are quite potent when compared with chemical carcinogens. Chromosome aberrations in cultured human peripheral lymphocytes have been demonstrated at Rn-222 alpha doses of about 48 mrads/y with an external gamma dose of about 100 mrads/y (Po77). Use of the dose conversion factor of these same investigators (Fi71) translates to a continuous exposure of about 0.042 pg/m^3 of Rn-222 and its daughters. Moreover, studies of underground miners have demonstrated significant increases in the incidence of lung cancer at 50 cumulative working level months of Rn-222 exposure occurring across a 17-year average period of exposure.* This is equivalent to about 0.1 of the working level of Rn-222 and its daughters in residential atmospheres. An equivalent air concentration would be about 20 nCi/m^3 of Rn-222 or 0.130 pg/m^3 of Rn-222 and its daughters. (For a more detailed discussion of working level exposures, see Chapter 8.)

3.2 Evidence That Radiation is Mutagenic

Radiation can change the structure, number, or genetic content of the chromosomes in a cell nucleus. These genetic radiation effects are classified as either gene mutations or chromosomal aberrations. Gene mutations refer to alterations of the basic units of heredity, the genes. Chromosomal aberrations refer to changes in the normal number or structure of chromosomes. Both gene mutation and chromosomal aberrations are heritable; therefore, they are considered together as genetic effects. Mutations and chromosomal aberrations can occur in somatic (body) or germ (reproductive) cells. In the case of germ cells, the mutagenic effect of radiation is not seen in those persons exposed to the radiation, but in their descendents.

Mutations often result in miscarriages or produce such undesirable changes in a population as congenital malformations that result in mental or physical defects. Mutations occur in many types of cells; no tendency toward any specific locus or chromosome has been identified. For this reason, they can affect any characteristic of a species. A relatively wide array of chromosome aberrations occur in both humans and animals.

Early experimental studies showed that x-radiation is mutagenic. In 1927, H. J. Muller reported radiation-induced genetic changes were reported in animals, and in 1928, L. J. Stadler reported such changes in plants (Ki62). Although genetic studies were carried out in the 1930's, mostly in plants and fruit flies (Drosophila), the bulk of the studies on mammals started after the use of nuclear weapons in World War II (UNSCEAR58).

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*Personal communication from E. P. Radford to Dr. Neal Nelson (ORP), 1981.

Very little quantitative data are available on radiogenic mutations in humans, particularly from low-dose exposures, for the following reasons: these mutations are interspersed over many generations, some are so mild they are not noticeable, and some mutagenic defects that do occur are similar to nonmutagenic effects and are therefore not necessarily recorded as mutations. The bulk of data supporting the mutagenic character of ionizing radiation comes from extensive studies of experimental animals, mostly mice (UNSCEAR77,82; NAS72,80). These studies have demonstrated all forms of radiation mutagenesis--lethal mutations, translocations, inversions, nondisjunction, point mutations, etc. Mutation rates calculated from these studies are extrapolated to humans (because the basic mechanisms of mutations are believed to be the same in all cells) and form the basis for estimating the genetic impact of ionizing radiation on humans (NAS80, UNSCEAR82). The vast majority of the demonstrated mutations in human germ cells contribute to both increased mortality and illness (NAS80, UNSCEAR82). Moreover, the radiation protection community is generally in agreement that the probability of inducing genetic changes increases linearly with dose and that no "threshold" dose is required to initiate heritable damage to germ cells.

A considerable body of evidence has been documented concerning the production of mutations in cultured cells exposed to radiation. Such mutations have been produced in Chinese hamster ovary cells, mouse lymphoma cells, human diploid fibroblasts, and human blood lymphocytes. Many of the radiation-induced specific types of mutations produced in human and Chinese hamster cultured cells are associated with structural changes in the X chromosome. Evidence suggests that these mutations may be largely due to deletions in the chromosomes. Thacker, Stretch, and Stephens found that human, mouse, and Chinese hamster cells all exhibit the same fixed probability of radiation-induced mutations (Th77). Analysis of published data on x- or gamma radiation-induced mutations in cultured cells of humans and mice show that when the induced mutation frequencies are plotted against log of survival, the relationship is linear. This relationship suggests that mutation frequency curves can be predicted from a knowledge of survival curves for each cell type.

Mutagenicity in human somatic cells has been demonstrated on the basis of chromosome aberrations detected in cultured lymphocytes. Chromosome aberrations in humans have been demonstrated in lymphocytes cultured from persons exposed to ingested Sr-90 and Ra-226 (Tu63); inhaled/ingested Rn-222, U-nat, or Pu-239 (Br77); or inhaled Rn-222 (Po78); and in atomic bomb survivors (Aw78). Although no evidence of health impact currently exists, these chromosome aberrations demonstrate that mutagenesis is occurring in somatic cells of humans exposed to ionizing radiation.

Evidence of mutagenesis in human germ cells (cells of the ovary or testis) is less conclusive. Studies have been made of several populations exposed to medical radiation, atomic bomb survivors, and a population in an area of high background radiation in India (UNSCEAR77). Although these studies suggest an increased incidence of chromosomal aberrations in germ cells following exposure to ionizing radiation, the data are not convincing (UNSCEAR77).

Investigators who analyzed the data on children born to survivors of the atomic bombings of Hiroshima and Nagasaki found no statistically significant genetic effects due to parental exposure (Sc81). They did find, however, that the observed effects are in the direction of genetic damage from the bomb radiation exposure. They also were able to calculate that an average doubling dose* of 156 rems of ionizing radiation will produce a 100 percent increase over the spontaneous mutation rate. The average doubling dose in mice is generally estimated to be much lower, about 30 to 40 rems. These doses apply to acute radiation exposure. Extensive experiments with mice indicate that the genetic yield from low-level, chronic exposures to radiation is about one-third that of acute radiation (Sc81). In a later report, the same researchers estimated an acute doubling dose of 250 rems (Sa82).

The incidence of serious genetic disease due to mutations and chromosome aberrations induced by radiation is referred to as genetic detriment. Serious genetic disease includes inherited ill health, handicaps, or disabilities. Genetic disease may be manifest at birth or may not become evident until some time in adulthood.

Researchers have attempted to measure genetic detriment due to radiation exposure by using indices such as years of life lost, relative length of hospitilization or medical care necessary, or time lost from work. Measures of genetic detriment have several shortcomings. For example, they do not differentiate with regard to the range of severities of a disease; nor do they include a measure of the impact of a disease on the family, health care centers, schools, and society in general. For example, measures of genetic detriment based on years of life lost is much higher for Down's syndrome than for Huntington's disease, largely because of the much higher incidence of Down's syndrome. The difficulty experienced by the families of those suffering from each genetic disease is not accounted for, however. Those genetic diseases that necessitate long-term stays in institutions may pose burdens on society that are inversely related to mortality.

Carter and the U.N. Committee (Ca80,82; UNSCEAR82) have provided approximate estimates of genetic detriment in a developed country. As shown in Table 3-1, dominant genetic diseases usually rank relatively low because their onset is late in life.

Using utilization of hospital services as an index of genetic detriment, researchers have found that children with dominant or recessive diseases or congenital malformations are, on the average, admitted to hospitals 5 to 7 times more often in their first year of life (Tr77). Children with any of these three types of genetic diseases spend considerably more time in the hospital than other children.

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* A doubling dose is one that will produce a 100 percent increase over the spontaneous mutation rate.

Radiation-induced genetic detriment thus includes impairment of life, shortened life span, and increased hospitalization. Only estimates of the frequency of radiation-induced genetic impairment are presented in Chapter 8 of this document. Although the numbers represent rough approximations, they are relatively small in comparison with the magnitude of detriment associated with spontaneously arising genetic diseases (UNSCEAR82).

Table 3-1. Estimates of genetic detriment in a developed country (UNSCEAR82)

Criteria for genetic determinant	Genetic diseases, listed in the order of severity (greatest to least)
Years of impaired life	Chromosomal X-linked Recessive Dominant Irregularly inherited
Years of life lost	Recessive Irregularly inherited X-linked Dominant
Degree of life impairment	Recessive Chromosomal X-linked Dominant
Impaired life weighted for degree of impairment	Recessive Chromosomal X-linked Dominant

3.3 Evidence That Radiation Is Teratogenic

Teratogenicity is the malformation of cells, tissues, or organs of a fetus resulting from physiologic and biochemical changes. Radiation is a well-known teratogenic agent. Case reports of radiation-induced teratology were made as early as 1921 (Sta21). By 1929, an extensive review of a series of pregnancies yielded data indicated that 18 of the children born to 76 irradiated mothers had abnormally small heads (microcephally) (Mu30). Although the radiation dose in these cases is not known, it was high.

Early experimental studies (primarily in the 1940's and 1950's) demonstrated the teratogenic properties of x-rays in fish, amphibia, chick, mouse, and rat embryos (Ru53). These experiments showed that the developing fetus is much more sensitive to radiation than the mother and provided data on periods of special sensitivity and dose-response. The malformations produced in the embryo depend on which cells, tissues, or organs in the fetus are most actively differentiating at the time of radiation. Embryos are relatively resistant to radiation-induced teratogenic effects during the earliest stages of their development, and are most sensitive during development of the neuroblast (these cells eventually become the nerve cells). These experiments showed that different malformations could be elicited by irradiating the fetus at specific times during its development.

Substantial evidence points to the ability of radiation to induce teratogenic effects in human embryos as well. In a recent study of mental retardation in children exposed _in utero_ to atomic bomb radiation in Hiroshima and Nagasaki, researchers found that damage to the child appears to be related linearly to the radiation dose that the fetus receives (Ot84). The greatest risk of damage occurs at 8 to 15 weeks, which is the time the nervous system is undergoing the most rapid differentiation and proliferation of cells. They concluded that the age of the fetus at the time of exposure is the most important factor in determining the extent and type of damage from radiation. A numerical estimate of mental retardation risk due to radiation is given in Chapter 8.

3.4 Uncertainties

Although much is known about radiation dose-effect relationships at high-level doses, uncertainty exists when dose-effect relationships based on direct observations are extrapolated to lower doses, particularly when the dose rates are low. As described in Chapter 8, the range of extrapolation varies depending on the sensitivity of the organ system. For breast cancer, this may be as small as a factor of four. Uncertainties in the dose-effect relationships are recognized to relate to such factors as differences in quality and type of radiation, total dose, dose distribution, dose rate, and radiosensitivity (including repair mechanisms, sex, variations in age, organ, and state of health). The range of uncertainty in the estimates of radiation risk is examined in some detail in Chapter 8.

The uncertainties in the details of mechanisms of carcinogenesis, mutagenesis, and teratogenesis make it necessary to rely on the considered judgments of experts on the biological effects of ionizing radiation. These findings, which are well documented in publications by the National Academy of Sciences and the United Nations Scientific Committee on the Effects of Atomic Radiation, are used by advisory bodies such as the International Council on Radiation Protection and Measurements (ICRP) in developing their recommendations. The EPA has considered all such findings in formulating its estimate of the relationship between radiation dose and response.

Estimates of the risk from ionizing radiation are often limited to fatal cancers and genetic effects. Quantitative data on the incidence of nonfatal radiogenic cancers are sparse, and the current practice is to assume that the total cancer incidence resulting from whole-body exposure is 1.5 to 2.0 times the mortality. In 1980, the NAS-BEIR Committee estimated the effects of ionizing radiation directly from epidemiology studies on the basis of both cancer incidence and the number of fatal cancers induced per unit dose (NAS80). The lifetime risk from chronic exposure can be estimated from these data, either on the basis of (1) relative risk (i.e., the percentage of increase in fatal cancer), or (2) absolute risk (i.e., the number of excess cancers per year at risk following exposure). The latter method results in numerically smaller estimated risks for common cancers, but a larger estimated risk for rare cancers.

3.5 Summary of Evidence That Radiation is a Carcinogen, Mutagen, and Teratogen

Radiation has been shown to be a carcinogen, a mutagen, and a teratogen. At sufficiently high doses, radiation acts as a complete carcinogen, serving as both initiator and promoter. With proper choice of radiation dose and exposure schedule, cancers can be induced in nearly any tissue or organ in both humans and animals. At lower doses, radiation produces a delayed response in the form of increased incidence of cancer long after the exposure period. This has been documented extensively in both humans and animals. Human data are extensive and include atomic bomb survivors, many types of radiation-treated patients, underground miners, and radium dial workers. Animal data include demonstrations in many mammalian species and in mammalian tissue cultures. A significant finding from tissue culture studies is that radiation induces cancers by a process that is similar to that of chemical carcinogens. Further, DNA altered by radiation can cause transformation of other cultured cells when introduced to normal cells, even when the change in the DNA is very small.

Evidence of mutagenic properties of radiation comes mostly from animal data, in which all forms of radiation-induced mutations have been demonstrated, mostly in mice. Tissue cultures of human lymphocytes have also shown radiation-induced mutations. Data on humans are less conclusive; however, estimates of genetic detriment due to radiation exposure have been made by the use of measures such as years of life lost or years requiring hospitalization.

Evidence that radiation is a teratogen has been demonstrated in animals and in humans. A fetus is most sensitive to radiation during the early stages of organ development (between 8 and 15 weeks for the human fetus). The radiation-induced malformations produced depend on which cells are most actively differentiating.

In conclusion, evidence of the carcinogenic, mutagenic, and teratogenic properties of radiation is very substantial. These health effects pose a detrimental risk to exposed persons.

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Chapter 4: EMISSION OF RADIONUCLIDES INTO THE AIR

4.1 Introduction

Radionuclides are used or produced in thousands of locations throughout the United States, including national defense weaponry facilities, nuclear powerplants, industrial plants, research and development laboratories, and medical facilities. Fossil-fuel combustion processes, such as large coal-fired boilers, make some contribution to the exposure of the general public. Certain kinds of mining and milling also substantially increase the local concentration of radionuclides in the air.

Although air cleaning equipment is usually used in these facilities, some radionuclides are still released into the air and can disperse into populated areas.

Sources of emissions of radionuclides to the air can be divided into the following groups:

(1) Department of Energy facilities

(2) Nuclear Regulatory Commission licensed facilities (exclusive of commercial nuclear power generating facilities) and non-DOE Federal facilities

(3) Coal-fired utility and industrial boilers

(4) Underground uranium mines

(5) Phosphate rock processing and wet-process fertilizer plants

(6) Elemental phosphorus plants

(7) Other mineral extraction and processing facilities

(8) Uranium fuel cycle facilities, including uranium mill tailing and high-level waste disposal facilities and commercial nuclear power generating facilities

(9) Low-energy accelerators.

These sources are described in the chapter, including the physical characteristics of the radionuclide releases (i.e., particle size and physical state) and the amount of release. This information is used in subsequent chapters to evaluate the movement of radionuclides through the environment.

Because of the large number of facilities within certain source categories (e.g., coal-fired industrial boilers), conducting a risk assessment for each plant is not practicable. Therefore, in some cases, it was necessary to develop a model or reference facility upon which to base a risk assessment. The reference facilities were developed from data obtained from multiple facilities within a source category. These data reflect the range in operating parameters and radionuclide emissions that are representative of the particular source category. The operating parameters and radionuclide emission rates of those source categories for which reference facilities were developed are discussed in the following subsections of this chapter.

4.2 Sources of Radionuclide Releases into the Air

Naturally occurring and manmade radionuclides are emitted to air from a variety of sources. Sources of manmade radionuclides include nuclear powerplants and other facilities that use nuclear fuel, research and development laboratories, medical facilities, and national defense facilities. The type and quantity of radionuclide emissions from these sources are typically well defined. Mining, mineral processing, and fossil-fuel combustion are also potential sources of naturally occurring radionuclides.

The discussions of the radionuclide emission sources provided in the following subsections include descriptions of the various facilities, specific emission release points (or areas), and a general description of the types of emissions. The quantities emitted are provided in Section 4.3.

4.2.1 Department of Energy (DOE) Facilities

The DOE owns or directs under contract many facilities that emit radionuclides into the air. The largest of these facilities and their locations are listed in Table 4.2-1. These facilities support weapons production and numerous research and development programs for the Department of Defense (DOD), including biomedical studies, studies of environmental and safety aspects of nuclear energy, and investigations concerning nuclear waste processing.

The diversity of operations among the various sites makes it difficult to assess DOE facilities on a generic basis. The major emissions from the various facilities, however, are similar and consist largely of inert gases such as argon (Ar-41), krypton (Kr-85 and 88), and xenon (Xe-133). These gases are heavier than air and only slightly soluble in water. Tritium (H-3) and oxygen (O-15) are also commonly emitted. A site-by-site review of each source follows. Volume II, Chapter 2, of this document discusses each of these facilities in greater detail.

Table 4.2-1. Department of Energy facilities

Laboratory	Location
Argonne National Laboratory	Argonne, Illinois
Brookhaven National Laboratory	Long Island, New York
Feed Materials Production Center	Fernald, Ohio
Fermi National Accelerator Laboratory	Batavia, Illinois
Hanford Reservation	Richland, Washington
Idaho National Engineering Laboratory	Upper Snake River, Plain, Idaho
Lawrence Livermore National Laboratory	Livermore, California
Los Alamos National Laboratory	Los Alamos, New Mexico
Oak Ridge Reservation	Oak Ridge, Tennessee
Paducah Gaseous Diffusion Plant	Paducah, Kentucky
Portsmouth Gaseous Diffusion Plant	Piketon, Ohio
Rocky Flats Plant	Jefferson County, Colorado
Savannah River Plant	Aiken, South Carolina
Ames Laboratory	Ames, Iowa
Bettis Atomic Power Laboratory	West Miflin, Pennsylvania
Knolls Atomic Power Laboratory	Schenectady, New York
Lawrence-Berkeley Laboratory	Berkeley, California
Mound Facility	Miamisburg, Ohio
Nevada Test Site	Nye County, Nevada
Pantex Plant	Amarillo, Texas
Pinellas Plant	Pinellas County, Florida
Rockwell International	Santa Susana, California
Sandia National Laboratories	Albuquerque, New Mexico
Stanford Linear Accelerator Center	Stanford, California
Reactive Metals, Inc.*	Ashtabula, Ohio

* See Volume II for description.

Argonne National Laboratory

Argonne National Laboratory is an energy research and development center that performs investigations in basic physics, chemistry, materials science, the environmental sciences, and biomedicine. Argonne also plays an important role as a nuclear and nonnuclear engineering center. The laboratory complex is located in Dupage County, Illinois, 43 kilometers southwest of Chicago.

Argonne National Laboratory has the following principal nuclear facilities:

(1) 10- and 200-kW research reactors

(2) A critical assembly reactor

(3) A 60-inch cyclotron

(4) A prototype, superconducting, heavy ion linear accelerator

(5) Van de Graaff and Dynamitron-type charged-particle accelerators

(6) A high-energy neutron source

(7) Cobalt-60 irradiation sources

(8) Laboratories engaged in work with multicurie quantities of the actinide elements

The 200-kW JANUS research reactor and the laboratory handling area (hot cells) are the main sources of radionuclide releases from the Argonne complex.

Specific details of the site activities and emissions are available from annual emission reports prepared by the laboratory (GO82), the DOE Effluent Information System (DOE81a), and environmental monitoring studies conducted by DOE (ERDA77a).

Brookhaven National Laboratory

Studies conducted at Brookhaven Laboratories pertain to the use, environmental effects, and transport of both nuclear and nonnuclear energy materials. Other research programs include applied nuclear studies involving various radioisotopes and investigations of the physical, chemical, and biological effects of radiation. Brookhaven Laboratory is located in the center of Long Island, about 113 kilometers from New York City.

The equipment and facilities used to support the research projects conducted at Brookhaven include several reactors, particle accelerators, and laboratories. Point and area sources of radionuclide releases at Brookhaven include:

(1) The 40-MW High-Flux Beam Reactor (HFBR)

(2) The Alternating Gradient Syncrotron, a proton accelerator used in ultra-high energy particle physics research

(3) The Brookhaven Linac Isotope Production Facility (BLIP)

(4) The Chemistry Linac Irradiation Facility (CLIF)

(5) The Brookhaven Medical Research Reactor

(6) The Van de Graaff accelerator

(7) Various chemistry and medical research laboratories

Most of the airborne radionuclide emissions from Brookhaven originate from the High-Flux Beam Reactor, the Brookhaven Linac Isotope Production Facility, and the Van de Graaff research generator. Lesser emissions are from the chemistry and medical research centers.

During 1981, emissions were identified from seven stacks, as listed in Table 4.2-2. Because very small quantities of radionuclides are released from the Hazardous Waste Management Area, the assessments of exposure and health risk at the Brookhaven site are based on airborne releases from the remaining six effluent stacks. Process descriptions, effluent data, and site information were obtained from reports prepared by Brookhaven Laboratories (Na82) and DOE studies (DOE81a, ERDA77a).

Table 4.2-2. Radionuclide emission points (stacks) at Brookhaven National Laboratories

Location	Stack height (m)
Brookhaven Linac Isotope Production Facility, Building-931	46
High-Flux Beam Reactor Hot Laboratory	98
Hazardous Waste Management Area	10
Medical Research Reactor Building-491	Unknown
Chemistry Building-555	Unknown
Medical Research Center	Unknown
Van de Graaff Accelerator Building-901	18

Fermi National Accelerator Laboratory

The Fermi National Accelerator Laboratory is principally involved with basic research in high-energy physics. Another important activity involves the treatment of cancer patients with neutrons released by the second stage of the accelerator. The Fermi complex is located east of Batavia, Illinois, in the greater Chicago area.

The accelerator at the Fermi Laboratory, a proton synchrotron, routinely operates at energies up to 400 GeV (billion electron volts). The proton beams produced in the accelerator are used in three different onsite experimental facilities: (1) the Meson area, (2) the Neutrino area, and (3) the Proton area. Production of radionuclides in these areas and by the accelerator occurs when either the proton beam itself or secondary particles interact with air.

Another source of radionuclides at Fermi Laboratory is a magnet-debonding oven, where failed magnets for the accelerator are baked at high temperatures to break down the adhesives that help form the magnets.

Hanford Reservation

The Hanford Reservation was established in 1943 as a plutonium production facility for nuclear armaments. Information used to evaluate the facility was obtained from DOE and Hanford reports (DOE81a, ERDA75, ERDA77b, Su82). Plutonium production has decreased, and other programs filled the gap, such as management and storage of radioactive wastes, reactor operations, fuel fabrication, energy research and development, and biophysical and biomedical research. The reservation, which is located 270 kilometers south of Seattle, Washington, is separated into four areas, which are designated the 100, 200, 300, and 400 Areas. The activities of each area are described briefly.

100 Area. The 100 Area contains the nine plutonium production reactors for which the site was originally developed. Eight of these reactors are currently on standby. Operating facilities include the N-Reactor and the 1706 Laboratory, which provides support services for the reactor.

200 Area. Activities conducted in the 200 Area include fuel processing, nuclear waste treatment and storage, equipment decontamination, and research. Plutonium reclamation from spent fuel is performed at the PUREX Plant in this area.

300 Area. The major facilities in the 300 Area are the Hanford Engineering Development Laboratory, the fuel fabrication facility, and the Life Sciences Laboratory. The Hanford Engineering Development Laboratory, the largest operation in this area, supports all activities of the development program for the fast breeder reactor. Life science research in this area includes plutonium inhalation studies and other programs investigating the physiological effects of radioactive materials.

400 Area. The only facility currently in operation in the 400 Area is the Fast Flux Test Facility. When the Fuel Materials Examination Facility currently under construction is completed, the 400 Area will be the center of the Hanford breeder reactor research program.

Idaho National Engineering Laboratory

The Idaho National Engineering Laboratory is a reactor testing facility in southeastern Idaho, about 56 kilometers west of Idaho Falls. The following four contractors operate facilities here: EG&G Idaho, Inc.; Allied Chemical Corporation; Argonne National Laboratory; and Westinghouse Electric Corporation.

EG&G Facilities. EG&G, Inc., operates several test reactors. These reactors provide operating information for the development of reactor safety programs, for determination of the performance of reactor materials and equipment, and occasionally, for use in research performed by private organizations. Other activities include disassembly and reassembly of large radioactive reactor components, preparation of test specimens for use in various operating reactors, and waste handling.

Allied Chemical Corp., Idaho Chemical Processing Plant. Fuel processing is the major operation that Allied conducts at this site. The Idaho Chemical Processing Plant stores irradiated fuel and reprocessed fuel, and converts high-level-radioactive liquid waste to solid form.

Argonne National Laboratory, West Facility. The Argonne National Laboratory complex currently has five operational facilities: the Experimental Breeder Reactor, the Transient Reactor Test Facility, the Zero Power Plutonium Reactor, the Hot Fuels Examination Facility, and the Laboratory and Office Support Complex. Each of these facilities provides research and physical support for the DOE fast breeder reactor project.

Westinghouse Electric Corporation. Westinghouse operates the Naval Reactor Facility at the Idaho Laboratory. This facility serves as a testing area for prototype naval reactors and as a disassembly and inspection area for expended reactor cores (DOE81a, DOE82a, ERDA77a, ERDA77c).

Lawrence Livermore National Laboratory

The Lawrence Livermore National Laboratory, situated 64 kilometers east of San Francisco, California, is primarily a nuclear weapons research and development center. Other activities, however, include research programs in laser isotope separation, laser fusion, magnetic fusion, biomedical studies, and nonnuclear energy.

Two accelerators, the Insulated Core Transfer Accelerator and the Electron Positron Linear Accelerator, are used in support of the fusion and neutron physics research programs. The Light Isotope Handling Facility supports research in the area of light isotopes. The remaining facilities at this site deal with equipment decontamination and waste disposal (DOE81a, UC82).

Los Alamos National Laboratory

Los Alamos National Laboratory is one of the prime research and development centers for DOE's nuclear weapons program. This facility is located about 100 kilometers north-northeast of Albuquerque, New Mexico. In addition to defense-related activities, programs include research in the physical sciences, energy resources, environmental studies, and biomedical applications of radiation.

Radionuclides are released from 13 technical areas at this site. These areas contain research reactors that produce materials for use in high-temperature chemistry applications, weapons systems development, nuclear safety program development, accelerator operations, biomedical research, development of isotope separation processes, and waste disposal (DOE81a, LANL82).

Oak Ridge Reservation

Oak Ridge National Laboratory, located about 35 kilometers west of Knoxville, Tennessee, is a multidisciplinary research facility that conducts basic and applied research into all aspects of energy production. Three major facilities are located on the Oak Ridge Reservation: Oak Ridge National Laboratory, Oak Ridge Gaseous Diffusion Plant, and the Y-12 plant.

The equipment facilities used to support research activities at Oak Ridge Laboratory include nuclear reactors, chemical pilot plants, research laboratories, and waste disposal and handling areas. Radionuclide emissions are released during isotope preparation and chemistry laboratory operations. Emissions from the Gaseous Diffusion Plant and Y-12 plant generally consist of particulates released during fuel processing and enrichment (DOE81a, EPA79a, UCC82a).

Paducah Gaseous Diffusion Plant

The DOE operation at the Paducah Gaseous Diffusion Plant consists of a uranium enrichment facility and a uranium hexafluoride manufacturing complex. The plant is located 6 kilometers south of the Ohio River in McCrasken County, Kentucky.

The primary activity at this site is the diffusion cascade for the enrichment of uranium in fissionable uranium-235 content. All stages of the enrichment cascade take place in five buildings on the site. The manufacturing facility produces uranium hexafluoride from uranium oxide feedstocks (DOE81a, UCC82b).

Portsmouth Gaseous Diffusion Plant

The Portsmouth Gaseous Diffusion Plant, situated in Pike County, Ohio, about 1.6 kilometers east of the Scioto River, is operated by Goodyear Atomic Corporation. Its primary function is the production of enriched uranium. Operations at this plant are similar to those described for the Paducah Gaseous Diffusion Plant. The most significant release point, which accounts for about 84 percent of total emissions, is the X326 Top Purge Vent (DOE81a, EPA79a, GAC82).

Rocky Flats Plant

Activities at the Rocky Flats Plant, located in Jefferson County, Colorado, about 26 kilometers from Denver, are restricted to fabrication and assembly of components for nuclear weapons and the support of these operations.

Fabrication operations include reduction rolling, blanking, forming, and heat treating. Assembly operations include cleaning, brazing, marking, welding, weighing, matching, sampling, heating, and monitoring. Because of the toxicity of plutonium, all material-handling activities that involve plutonium are performed under strictly controlled conditions.

Solid residue generated during plutonium-related operations is recycled through one of two plutonium-recovery processes. Process selection depends on the purity and plutonium content of the residue. Both processes produce a plutonium nitrate solution from which the metal can be extracted. The recovered plutonium is returned to the storage vault for use in foundry operations. A secondary objective of the process is the recovery of americium-241.

Radionuclides are released from short stacks and building vents at this plant. Quantities are similar at several of the release points. Building 771, Main Plenum, was selected for comparison purposes and calculations. This point releases 54 percent of the plutonium-239 and -240 and 3 percent of the uranium-233, -234, and -235 emitted at Rocky Flats. The most significant release point for uranium is from a single duct in Building 883, which releases approximately 19 percent of the total uranium emissions from the plant (DOE81a, EPA79a).

Savannah River Plant

The facilities at the Savannah River Plant are used primarily to produce plutonium and tritium, the basic materials required for nuclear weapons. Materials for medical and space applications are also manufactured here, however. The Savannah River Plant is situated along the Savannah River at a site 35 kilometers southeast of Augusta, Georgia. The site covers about 770 square kilometers.

Operations are grouped into five major areas (designated the 100, 200, 300, 400, and 700 Areas) according to their operational function in the plutonium manufacture/recovery process.

100 Area - Nuclear Production Reactors. These production reactors are currently in operation; a fourth is being upgraded. The three operating reactors produce plutonium and tritium by irradiation of uranium and lithium. Heavy water is used both as a neutron moderator and as a primary coolant.

200 Area - Separations and Waste Management Facilities. Nuclear fuel reprocessing occurs in this area. Plutonium is recovered from irradiated uranium by the PUREX solvent-extraction process. Enriched uranium and plutonium-238 are recovered from other irradiated materials by a solvent-extraction procedure similar to the PUREX process.

300 Area - Fuel and Target Fabrication. Tubular fuel and target elements are produced by cladding depleted uranium fuel in aluminum or aluminum/lithium shells. A low-power reactor and a subcritical test reactor are then used to test for assembly defects.

400 Area - Heavy Water Production and Recovery. Heavy water is produced from river water by distillation and extraction. Heavy water is also recovered from contaminated reactor coolant. Heavy water is transported from this area to the 100 Area for use in the production reactors.

700 Area - The Savannah River Laboratory. Research and process development work is performed at the Savannah River Laboratory. Major activities in this area include fabrication of fuel element and target prototypes; fabrication of radioisotopic sources for medical, space, and industrial applications; thermal and safety studies of reactor operations; and applied research in the areas of physics and the environmental sciences (DOE81a, DOE81b).

Feed Material Production Center

The Feed Material Production Center, located 32 kilometers northwest of Cincinnati, Ohio, produces uranium metal and other materials for DOE facilities. The uranium may be natural, depleted, or enriched with respect to uranium-235.

Raw materials are processed in the following manner. The material is first dissolved in nitric acid and separated by liquid organic extraction. The recovered uranium is reconverted to uranyl nitrate, heated to form uranium trioxide, reduced to uranium dioxide with hydrogen, and reacted with hydrogen fluoride to form uranium tetrafluoride. Purified metal is made by reacting the uranium tetrafluoride with metalic magnesium in a refractory-lined vessel (DOE81a, EPA79a, ERDA77d).

Ames Laboratory

Until 1978, the Ames Laboratory, which is operated by Iowa State University, was used as a neutron source for the production of byproduct materials and the neutron irradiation of various materials for research. The reactor was fueled with enriched uranium, moderated and cooled by heavy water (D_2O), and operated continuously at 5000 watts thermal. Operation of the Ames Laboratory Research Reactor was terminated on December 1, 1977. Decommissioning began January 3, 1978, and was completed on October 31, 1981. A waste processing and disposal facility that is still located at the site serves the campus reactor and research laboratories.

Prior to its decommissioning, the major airborne releases from the research reactor were tritium and argon-41. Tritium, the major radionuclide released during the 1981 decommissioning activities, was emitted from the 30-meter reactor stack, which is 215 meters from the nearest property boundary. Monitoring has indicated that no airborne emissions from the research laboratories have reached the main campus (EPA79a, DOE81a, DOE82b, ERDA77e).

Bettis Atomic Power Laboratory

The Bettis Atomic Power Laboratory is situated on an 0.8-square-kilometer tract in West Mifflin, Pennsylvania, approximately 12 kilometers south of Pittsburgh. This facility designs and develops nuclear power reactors. The most significant program currently in progress is the fabrication of fuel assemblies for DOE's light-water breeder reactor program (BAPL82, DOE81a, DOE82c, ERDA77a).

Knolls Atomic Power Laboratory

Knolls Atomic Power Laboratory has facilities at three separate sites: Knolls, Kesselring, and Windsor. Development of nuclear reactors and training of operating personnel are the major efforts at the Knolls Laboratory. The Knolls and Kesselring complexes are located near Schenectady, New York, and the Windsor site is near Windsor, Connecticut. Pressurized water reactors are located at both Kesselring and Windsor sites, where operating personnel are trained.

All releases of radionuclides from the Knolls sites are from elevated stacks (DOE81a, DOE82c, ERDA77a).

Lawrence-Berkeley Laboratory

The research facilities of Lawrence-Berkeley Laboratory are located on the Berkeley campus of the University of California. These facilities include four large accelerators, several small accelerators, several radiochemical laboratories, and the Tritium Labeling Laboratory. The large accelerators include the Bevatron, the Super HILAC, the 224-centimeter Sector-Focused Cyclotron, and the 467-centimeter Cyclotron.

The Tritium Facility was designed to accommodate kilocurie quantities of tritium as a labeling agent for chemical and biomedical research. Radiochemical and radiobiological studies in many laboratories typically use millicurie quantities of various radionuclides (DOE81a, LBL81).

Mound Facility

The Mound Facility, located in Miamisburg, Ohio, about 16 kilometers southwest of Dayton, Ohio, has a variety of active programs. These include research and development, processing of solid wastes for tritium recovery, fabrication and testing of weapons components, production of stable isotopes for the market, and manufacture of radioisotopic heat sources for military and aerospace applications.

The principal emissions of tritium and plutonium emanate from nine buildings, designated as HH, SW, H, PP, R, SM, WD, WDA, and 41. Buildings HH and SW, which contain the tritium recovery and reprocessing facilities, are the sole release points of tritium. Plutonium is released from the other facilities as a result of heat source production and waste disposal operations (DOE81a, EPA79a, Fa82).

Nevada Test Site

The Nevada Test Site lies about 100 kilometers northwest of Las Vegas, Nevada, in Nye County. This facility, which is part of DOE's weapons research and development complex, is responsible for design, maintenance, and testing of nuclear weapons. Other activities at this site include development of new nuclear energy technologies and radioactive waste disposal.

Radionuclide emissions result primarily from underground tests of nuclear weapons. Sources of these releases include drill-back operations, tunnel ventilation, leakage of gases from underground test sites, and resuspension of contaminated soils (DOE81a, ERDA77a, ERDA77f).

Pantex Plant

The Pantex Plant, located 30 kilometers northeast of Amarillo, Texas, is a nuclear weapons assembly and disassembly plant. Because most radioactive materials handled during the assembly of nuclear weapons are contained in sealed vessels, normal operations involving these materials do not result in major releases of radionuclides (DOE81a, DOE82c, ERDA77a, Mab82).

Pinellas Plant

The Pinellas Plant, located 10 kilometers northwest of St. Petersburg, Florida, is a major facility engaged in the production of nuclear weapons. Although descriptions of the principal operations resulting in atmospheric releases of radioactive materials could not be found in the literature, they are neutron generator development and production, testing, and laboratory operations. Small, sealed, plutonium capsules are used as heat sources in the manufacture of radioisotopic thermoelectric generators. The heat sources are triple-encapsulated to prevent release of plutonium to the atmosphere.

Emissions of radionuclides were identified from three sources: the Main stack, Laboratory stack, and Building stack (DOE81a, EPA79a).

Rockwell International

Rockwell International operates two facilities, one near Los Angeles and one near Santa Susana, California. These facilities conduct research and development and also manufacture nuclear reactor components. The Los Angeles facility performs uranium fuel processing operations and conducts research involving gamma radiation. The Santa Susana facility uses neutron radiography to inspect nuclear reactor components. This facility also serves as a materials handling laboratory and waste processing operation for other DOE facilities.

Radionuclide emissions originate from the materials handling laboratory and the waste processing facilities at the Santa Susana site (DOE81a, EPA79a, ESG82).

Sandia National Laboratories

The operations at Sandia National Laboratories near Albuquerque, New Mexico, include weapons testing, arming and fusing nuclear weapons, and developing modifications to delivery systems. The major facilities include the Sandia Pulsed Reactor, the Annular Core Pulsed Reactor (both of which are used to irradiate test materials), and the Relativistic Electron Beam Accelerator. Support facilities include the Neutron Generator Facility, the Tube Loading Facility, the Fusion Target Loading Facility, the Tritium Laboratory, and the Nondestructive Test Facility, all of which are located in Technical Areas (TA) I and V. TA-I, in the northwest corner of the site, also houses research and design laboratories. Technical Area III is the site of the Sandia low-level radioactive waste dump (DOE81a, DOE82c, ERDA77a, SNL82).

Stanford Linear Accelerator Center

The Stanford Linear Accelerator Center is a large research laboratory devoted to theoretical and experimental research in high-energy physics and to the development of new techniques in high-energy accelerator particle detectors. This accelerator complex is located about halfway between San Francisco and San Jose, California. The main tool of the laboratory is a linear accelerator that is used to accelerate electrons and positrons. Two storage ring facilities, SPEAR and PEP, are used to generate high-energy particles by the collision of the opposing particle beams. Colliding beam storage rings such as SPEAR and PEP truly "recycle" the beam particles. The same particles are brought into collision over and over again, rather than striking a target only once. For this reason, colliding-beam devices generate much less radiation and residual radioactivity than do conventional accelerators.

No immediate venting of the accelerator facility occurs. A waiting period allows all isotopes (with the exception of argon-41) to decay before they are exhausted from the facility. Therefore, the release of radioactivity is infrequent and limited to argon-41 for brief periods of 30 to 60 minutes. Airborne releases are from the accelerator vent stack (DOE81a, DOE81c).

4.2.2 Nuclear Regulatory Commission (NRC) Licensed Facilities and non-DOE Federal Facilities

This source category encompasses six different classifications of facilities: Research and Test Reactors, Accelerators, Radiopharmaceutical Manufacturing, Department of Defense Activities, Radiation Source Manufacturing, and other NRC Licensees. These facility classifications include sources licensed by NRC and sources licensed by States under an agreement with NRC. Most of the emissions are gases containing isotypes of argon, hydrogen, oxygen, nitrogen, krypton, and xenon. Small amounts of iodine and technetium occur from radiopharmaceutical applications. These sources are discussed in more detail in Volume II, Chapter 3. Brief discussions of the characteristics of the major sources follow.

Research and Test Reactors

This category consists of land-based, NRC-licensed reactors that are operated for purposes other than commercial power production, i.e., for basic and applied research and for teaching. Seventy such reactors are currently licensed to operate in the United States. Design types include heavy water, graphite, tank, pool, homogeneous solid, and uranium-zirconium hydride. These reactors are operated to test reactor designs; to test reactor components and safety features; to support basic and applied research in the fields of physics, biology, and chemistry; and to educate. Power levels range from near zero to 10 MW.

Airborne emissions from research and test reactors are usually limited to argon-41 and tritium. Because the emissions from each of the facilities in this classification generally vary only in the quantity of radionuclides emitted, a reference facility was chosen to be representative of the entire group for the purpose of determining emission characteristics for dose and health risk assessments.

The reference facility used in the risk assessments was a university heavy-water reflected reactor. This reactor can achieve a power level of 5 MW with atmospheric emissions equal to the highest levels reported to the NRC (NRC77, Ki79). A stack height of 50 meters was used for assessments of the radionuclide releases.

Radiopharmaceutical Industry

The use of radioactive materials in medical treatments and research has been steadily increasing. Manufacturers/suppliers, users, and waste receivers are sources of radionuclide emissions from this source category.

Manufacturers/Suppliers. Radiopharmaceutical manufacturing involves numerous chemical processes with the potential for releasing radioactive materials into the air. Most materials used in the manufacture of radiopharmaceuticals are produced in nuclear reactors. Reactor-produced materials account for up to 80 percent of the marketed pharmaceuticals.

In a reactor, the main steps in radionuclide production are as follows (EPA80):

(1) A suitable target is prepared and irradiated with neutrons

(2) For removal of undesirable impurities or for concentration of the desired product, irradiated targets are processed by dissolution or by more complicated processes, such as ion exchange, precipitation, or distillation.

(3) Radionuclides are placed in inventory, dispensed, and packaged for shipment.

Smaller amounts of certain radionuclides are also produced in particle accelerators, such as the cyclotron. Cyclotrons can be used to produce nuclides with decay characteristics that are preferable to those of other isotopes of the same element produced in reactors. Cyclotrons can also be used to produce isotopes of elements for which no reactor-produced nuclides exist.

The emissions from the reference facility represented the highest values reported. Airborne releases are from a single 15-meter-high stack (TRI79a, EPA79a, NRC81, Coa82, Fra82a, Fra82b, Le79, Ro82b).

Users. Radionuclides are used extensively for medical diagnosis, therapy, and research. The number of medical facilities using radioactive materials has grown from 38 in 1946 to more than 10,000 at the present time. With the exception of radioactive gases such as xenon, radionuclides are generally handled by hospitals in solid or liquid form, which tends to decrease the likelihood of airborne radionuclide releases.

For assessments of dose and health risk, a reference facility was designated as a typical effluent source for subsequent modeling. The parameters assigned to the reference facility are typical of a large hospital with nuclear medicine capabilities. Principal airborne releases are from building ventilation exhausts with an effective height of 10 meters (Le80, NRC81, Ro82a, TRI79a).

Waste Receiving Facilities

Most radioactive emissions from radiopharmaceutical users are released as liquids to sanitary sewer systems. When these liquid wastes are treated, radionuclides may be emitted into the air. Radionuclide releases at sewage treatment plants depend upon several factors. The chemical and physical properties of wastewater and sludge influence the potential amount of radioactivity released; for example, the potential for release is greater at points in the treatment process where wastewater pH is acidic. Other factors that affect radionuclide releases include decay losses, evaporative losses, solids removal, degree of system retention, and dilution.

Sludge drying and incineration are the greatest sources of radionuclide emissions from sewage treatment plants because the high temperatures employed in these processes (typically 725°C) volatilize the iodine and technetium. In addition, sludge incineration has the smallest time delay (compared with other sludge treatment processes) and the greatest potential for release of particulates due to mechanical agitation of ash and combustion gases in the incinerator (TRI79a). The selected reference treatment plant is one that dries and incinerates sludge because these activities release the most radionuclides.

Department of Defense Facilities

Facilities operated by the Department of Defense fall into three categories: the Armed Forces Radiobiology Research Institute, U.S. Army Reactors, and U.S. Navy Shipyards. The Radiobiology Research Institute and U.S. Army reactor facilities research the effects of radiation on health and on electronics used by the armed forces. U.S. Navy releases of airborne radionuclides are from reactors used for propulsion in the submarine and surface fleets.

Armed Forces Radiobiology Research Institute. The Armed Forces Radiobiology Research Institute is located on the grounds of the National Naval Medical Center in Bethesda, Maryland, approximately 20 kilometers northwest of Washington, D.C. In support of Department of Defense radiation research, the Institute operates a TRIGA Mark-F pool-type thermal research reactor and a linear accelerator (LINAC). Most of this research involves the medical effects of nuclear radiation and the effects of transient radiation on electronics and other equipment.

The Mark-F reactor is licensed by the NRC to operate at steady-state power levels up to 1.0 MW (thermal). This reactor is also capable of pulse operations. At peak power it can produce a 10-msec pulse of about 2500 MW (thermal). The Institute's linac typically operates in an energy range of 18 to 20 MeV, but it can operate at energies up to 30 MeV. Emissions from the reactor and accelerator are released from a common stack atop the Institute building (Sh81).

U.S. Army Facilities. The U.S. Army Test and Evaluation Command operates two reactors: the Army Pulse Radiation Facility at Aberdeen Proving Ground, Maryland, and the Fast Burst Reactor at White Sands Missile Range, New Mexico. These reactors are similar in design and are used to support Army and other Department of Defense (DOD) studies in nuclear radiation effects.

Both Army reactors are bare, unreflected, and unmoderated and they are fueled with enriched uranium. These reactors are capable of self-emitting, superprompt, critical-pulse operations, as well as steady-state operations at power levels up to 10 kW (EPA79a). The reactors are used primarily by DOD and defense contractors to study the effects of nuclear weapons on electronics and other DOD equipment.

The White Sands Laboratory is the principal source of radioactive airborne emissions from Army reactors. Concrete structures around the reactor at White Sands reflect, and thus lower, the energy of the neutrons from the reactor. These low-energy neutrons produce airborne radioactivity in the reactor building by activation of argon-40 in the air. Concrete structures at Aberdeen are farther from the reactor; hence, much less (essentially zero) argon-41 is produced at this facility because there are very few low-energy neutrons (TRI79a, AMTE81, Aa82, De76).

U.S. Navy Facilities. Almost all airborne emissions of radionuclides from U.S. Navy facilities result from activities at the nine naval shipyards (listed in Table 4.2-3), where construction, overhaul, refueling, and maintenance of the Navy's nuclear fleet of 133 submarines and ships take place. Operations performed at these shipyards include construction, startup testing, refueling, and maintenance of the pressurized water reactors that power the nuclear fleet. Radioactive wastes generated by these activities are processed and sealed at the shipyards and then shipped to commercial waste disposal sites.

Table 4.2-3. U.S. Naval Shipyard facilities

Facility	Location
Mare Island Naval Shipyard	Vallejo, California
Electric Boat Division, General Dynamics	Groton, Connecticut
Pearl Harbor Naval Shipyard	Pearl Harbor, Hawaii
Portsmouth Naval Shipyard	Kittery, Maine
Ingalls Shipbuilding Division	Pascagoula, Mississippi
U.S. Naval Station and Naval Shipyard	Charleston, South Carolina
Newport News Shipbuilding and Drydock	Newport News, Virginia
Norfolk Naval Shipyard	Portsmouth, Virginia
Puget Sound Naval Shipyard	Bremerton, Washington

The primary sources of airborne radioactive emissions from naval shipyards are the support facilities that process and package radioactive waste materials for shipment to disposal sites. These facilities handle solid, low-level radioactive wastes, such as contaminated rags, paper, filters, ion exchange resins, and scrap materials.

During operation, shipboard nuclear reactors release small amounts of radioactivity (carbon-14) into the atmosphere. Most of these releases take place at sea, more than 12 miles from shore (Ri82 and EPA77a).

Radiation Source Manufacturers

The term "radiation source" refers to radioactive material that is enclosed in a sealed container or other matrix that prevents its dispersion. Radiation sources are used in a wide variety of industrial and consumer products, including (1) radioisotope gauges, which measure the thickness of industrial products; (2) static eliminators, which are used to reduce static electricity in industrial machines; (3) nondestructive testing equipment; (4) self-illuminating signs and watch dials; and (5) smoke detectors (EPA79a).

Manufacturers of radiation sources process bulk quantities of radioactive materials received from radionuclide production facilities such as accelerators or reactors. During processing operations, the radioactive materials are handled with remote manipulators and custom-made enclosures, such as glove boxes (Cob83).

Other NRC Licenses (Cob83)

This category includes three different groups of facilities: NRC-licensed laboratories, low-level waste disposal sites, and NRC-licensed mineral and metal processing facilities.

Laboratories. The NRC-licensed laboratories include test, research, and development laboratories in industry, government agencies, and academic and research institutions. Approximately 700 laboratories are licensed by Agreement States to handle radioisotopes in an unsealed form. The EPA assumes that an equal number of NRC licensees handle unsealed radioisotopes.

Laboratory facilities at the various sites vary from a single small multipurpose laboratory up to 300 individual laboratories located within several buildings at a major university. Both academic and industrial laboratories use byproduct materials in basic research and development. Medical research laboratories conduct basic chemical and applied radionuclide research related to a broad spectrum of diseases and health problems. Government laboratories use radionuclides for specific purposes, such as food and drug testing, water and air quality measurements, and ocean and fisheries monitoring.

Waste Disposal Sites. Of the six commercial low-level radioactive waste disposal sites, only three are currently operational. These operational sites are located at Barnwell, South Carolina; Beatty, Nevada; and Richland, Washington.

The operational sites accept low-level radioactive wastes in a stabilized form, but not special nuclear materials, transuranics, or spent reactor fuels. Wastes accepted for disposal by shallow-land burial must meet specific site acceptance criteria. The disposal sites typically consist of a large fenced area of about 100 ha. Wastes are usually buried in the transport containers in which they arrive at the site to minimize atmospheric radioactive emissions.

Mineral and Metal Processing Facilities. Facilities that extract metals from thorium- and uranium-bearing ores are licensed by NRC or by an Agreement State. Six facilities, located in California, Florida, Illinois, New Mexico, and Pennsylvania (two facilities), are licensed by NRC; and four facilities, located in Alabama, Colorado, Oregon, and Tennessee, are licensed by Agreement States. At facilities licensed by NRC, columbium and tantalum followed by rare earth extraction processes are the principal sources of radioactive materials that require control under present provisions of 10 CFR 40.

Most Agreement-State- and NRC-licensed facilities are processing uranium- and thorium-bearing ores for refractory metals, their oxides, or rare earth metals. The industrial processes used in licensed facilities vary from wet chemical and solvent extraction to high-temperature sintering and smelting.

4.2.3 Coal-Fired Utility and Industrial Boilers

Coal-fired boilers are used to generate electricity, steam, and hot water for public and industrial consumption. Electric utility boilers are generally larger than industrial boilers, and their design range is also much more limited. Coal contains traces of naturally occurring radionuclides, which tend to be concentrated into the fly ash during combustion. The radionuclide emissions are in the form of fine insoluble particulate matter and consist largely of uranium, thorium, and their decay products. The quantity of radionuclides released into the atmosphere varies, depending on the radionuclide content of the coal, the furnace design, and the design and efficiency of the particulate control equipment.

In 1979 there were 1224 coal-fired utility boilers with a total generating capacity of 225 GW in service. By 1985 there will be approximately 1360 coal-fired units with a generating capacity of 307 GW in service (TRI79b). For an efficient assessment of the risks posed by radionuclide emissions from coal-fired utility boilers, a reference facility was developed. The reference facility is assumed to have a stack height of 185 meters and a plume rise of 50 meters, typical of large utility boilers.

Parameters for the reference industrial boiler were determined by the same general methods as those used for utility boilers, but they were based on lower thermal capacities and coal consumption. The reference industrial boiler facility is assumed to have a stack height of 150 meters.

4.2.4 Underground Uranium Mines

In 1982, underground uranium mining accounted for about 46 percent of all U.S. uranium production (DOE83). All U.S. uranium is currently mined in the West, more than 65 percent of it in New Mexico, Wyoming, and Texas.

A modified room-and-pillar method is generally used for underground uranium mining. A large-diameter main entry shaft is drilled to a level below the ore body, and a haulageway is established underneath the ore body. Vertical raises are then driven up from the haulageway to the ore body. Development drifts are driven along the base of the ore body connecting with the vertical raises. Mined ore is hauled along the development drifts to the vertical raises and is gravity-fed to the haulageway for transport to the main shaft for hoisting to the surface.

Ventilation shafts are installed at appropriate distances along the ore body, and typical ventilation flow rates are on the order of 6000 m^3/min. The principal radioactive effluent in the mine ventilation air is the large amount of gaseous radon-222 and is released during mining operations. Additional radon-222 and particulate (uranium and thorium) emissions result from surface operations at underground mines (EPA79a, EPA82d).

Radon is a heavy gas that is only slightly soluble in water. Because radon is a noble gas, it is chemically inactive. Radon cannot be scrubbed or filtered from an exhaust gas stream and generally does not adhere to particulate matter.

The major source of airborne radionuclides released from underground uranium mines is the ventilation air exhausted from the mine. Large underground mines usually have several vents, sometimes as many as 15, spread out over a large area. Emissions from these vents vary greatly and depend on factors such as the size of the working area, mining practices, production rate, ore grade, and ventilation rates (Ja80).

Several above-ground operations also may release radionuclides as a result of waste rock storage, wind erosion, and ore dumping and loading operations. These releases take the form of gas and particulate emissions (EPA83a).

For determination of the effect of radionuclide releases from underground uranium mines, a reference facility was composited from available site information for working mines (DOE83, EPA79a, Ja80). The parameters for the reference mine used for estimating emissions to the air are listed in Table 4.2-4. Emission data are presented in Section 4.3.

Table 4.2-4. Parameters of reference underground uranium mine

Parameter	Value
Ore grade	0.22% U_3O_8
Ore production	102,000 MT/y[a]
Days of operation	250 days/y
Number of vents	5
Vent height	3 meters
Radon emissions	11,000 Ci/y

[a] MT/y = metric tons/year

4.2.5 Phosphate Rock Processing and Wet-Process Fertilizer Plants

Mining of phosphate rock is the fifth largest mining industry in the United States. The southeastern United States is the center of phosphate mining operations; over 90 percent of the U.S. phosphate is mined in Florida, Tennessee, and North Carolina. Concentrations of uranium and its decay products in phosphate rock can be as much as 100 times greater than those in natural soil and rocks, and the handling and processing of this rock can release elevated concentrations of radionuclides in either gaseous or particulate form.

The as-mined phosphate rock is usually ground to a uniform size and dried. Both the drying and grinding operations can produce significant amounts of dust containing radionuclides. This step is frequently followed by calcining, which involves heating the rock to remove unwanted organics. This process step can release additional particulates as well as gases containing vaporized radionuclides.

Most phosphate rock produced in this manner is used in the production of agricultural fertilizers. This wet-process manufacture of fertilizer produces phosphoric acid from the phosphate rock, which is used to produce diammonium phosphate or triple superphosphate. Because this process involves further crushing and extensive handling of the phosphate rock, the radionuclides contained in the rock are released into the atmosphere as particulates (TRW82a).

The parameters of a reference phosphate-rock drying and grinding plant and a wet-process fertilizer plant are shown in Tables 4.2-5 and 4.2-6.

Table 4.2-5. Parameters of reference phosphate-rock drying and grinding plant

Parameter	Dryers	Grinders
Number of units(a)	3	4
Phosphate rock processing rate (MT/y)	2.7E+6	1.2E+6
Operating factor (h/y)	6570	6460
Uranium-238 content of phosphate rock (pCi/g)(b)	40	40
Stack parameters		
Height (meters)	20	20
Diameter (meters)	2	2
Exit gas velocity (m/sec)	10	10
Exit gas temperature (°C)	60	60
Type of control system	Low-energy scrubber	Medium-energy scrubber
Particulate emission rate [g/MT (lb/ton)]	130 (0.26)	25 (0.05)

(a) Dryer units process 145 MT/h; grinder units process 45 MT/h.

(b) Uranium-238 is assumed to be in equilibrium with its daughter products.

Table 4.2-6. Reference wet-process phosphate fertilizer plant

Parameter	Process	
	DAP[a]	GTSP[b]
Production rate (MT/y)	5.2E+5	2.7E+5
Operating factor (h/y)	8160	8160
Radionuclide content of product (pCi/g)[c]		
Uranium-238, uranium-234, thorium-230	60	60
Radium-226	5	20
Lead-210, polonium-210	30	30
Stack parameters		
Height (meters)	40	40
Diameter (meters)	2	2
Exit gas velocity (m/sec)	10	10
Exit gas temperature (°C)	60	60
Type of control system	Venturi scrubber	Venturi scrubber
Particulate emission rate (g/MT)	164	100

(a) DAP Diammonium phosphate.

(b) GTSP Granular triple superphosphate.

(c) Data from EPA78a.

4.2.6 Elemental Phosphorus Plants

Production of elemental phosphorus in the United States utilizes about 10 percent of all phosphate rock mined annually. Phosphorus is the principal feed material in the production of phosphate-based detergents, high-grade phosphoric acid, and organic chemicals. The phosphate rock used in the production of phosphorus contains greater-than-normal concentrations of uranium and its decay products. Releases of radionuclide particulates occur during processing of this feed material. Heating the phosphate rock to high temperatures during its processing also releases radionuclides into the air (An81a, An81b, TRI81). The major radionuclides emitted are polonium-210 and lead-210. The polonium and lead are emitted as fine particulate matter. Recent test data indicate that approximately 90 percent of the polonium-210 emissions have an aerodynamic diameter of less than 1.5 micrometers. Lead-210 emissions less than 1.5 micrometers in diameter ranged from about 70 percent to 90 percent.

The processing of phosphate-bearing rock into elemental phosphorus starts with crushing and sizing. This is followed by calcining, which burns away any organic material and allows the rock to be sorted into uniform sizes for further processing. The rock, along with other materials, is then fed into an electric furnace to produce phosphorus. The phosphorus leaves the furnace as a gas, which is cooled and collected in water condensers (EPA77b, EPA79b). The remaining gas is vented or recycled to the calciner.

The stack parameters used in the assessments of health risks from the elemental phosphorus plants are presented in Table 4.2-7.

Table 4.2-7. Calciner stack emission characteristics

Plant	Stack height (meters)	Heat emission (calories/sec)
FMC Pocatello, Idaho	30	8.8E+5
Monsanto Soda Springs, Idaho	31	2.0E+6
Monsanto Columbia, Tennessee	35	1.0E+6
Stauffer Silver Bow, Montana	27	3.0E+4
Stauffer Mt. Pleasant, Tennessee	35	6.0E+5
Occidental Columbia, Tennessee	31	1.2E+6

4.2.7 Mineral Extraction Industry Facilities

All industrial operations that are involved in the extraction and processing of mineral ores release some quantity of radionuclides into the atmosphere. The aluminum, copper, zinc, and lead industries have the greatest potential for radionuclide releases because of the high volume of material processed and because they all utilize high temperature smelting. These emissions are largely in the form of fine particles of uranium, lead, polonium, and (at times) thorium. Most of these radioactive metallic elements occur in the oxide and sulfate form.

Aluminum Industry Facilities

The production of aluminum differs somewhat from other mineral-extraction industries because contaminants in the ore are removed during the milling of the ore rather than during smelting. The aluminum ore (usually bauxite) is converted into aluminum oxide at the mines, and subsequently shipped to the smelters for final processing. Aluminum metal is produced in electric reduction cells. Particulate emissions from the process reflect the composition of the feed materials, which includes alumina, carbon, aluminum fluoride, and cryolite (EPA79c, EPA82a).

The parameters of a reference aluminum reduction plant are listed in Table 4.2-8. These values are used to estimate the radionuclide emissions to air.

Table 4.2-8. Parameters of reference aluminum reduction plant (TRI81)

Parameter	Value
Capacity	136,000 MT/y aluminum
Capacity factor	0.94
Type of equipment	Center-worked prebake cells
Main stack parameters	
Height	36 m (4 stacks)
Diameter	3 m
Exit gas velocity	80 m/sec
Exit gas temperature	160°C
Roof monitor	
Height	10 m
Width	1.2 m
Exit gas velocity	0.01 m/sec
Exit gas temperature	37°C
Anode bake plant	
Height	30 m
Diameter	1.8 m
Exit gas velocity	4.5 m/sec
Exit gas temperature	96°C

Copper Industry Facilities

Copper ore is processed to yield a concentrate containing copper, sulfur, iron, and other remaining impurities, which are removed by smelting. The three major steps in copper production are roasting, smelting, and converting. Each of these steps release sulfur oxides and particulates that may contain radionuclides.

The purpose of roasting the concentrated copper ore is to remove some of its sulfur content. Some particulate material is released during this process. All domestic copper smelters produce an intermediate grade of copper by smelting the copper ore at high temperatures with other materials to form two liquids that separate into a mixture of copper and iron impurities and a layer containing a significant fraction of the other materials in the ore. The converter process removes the iron impurities from the copper and iron mixture at high temperatures before its final purification in a refining furnace.

The parameters of a reference copper smelter that were used to estimate the radioactive emissions to the air are shown in Table 4.2-9. The copper output capacity of the reference plant is 56,000 MT/y, and a capacity factor of 0.75 was chosen for this plant. Main stack heights for facilities without roasters range from 61 to 228 meters. The control equipment applied to the reference facility was chosen to represent typical equipment on actual copper smelters (EPA82a).

Table 4.2-9. Parameters of reference copper smelter (TRI81)

Parameter	Value
Capacity	56,000 MT/y
Capacity factor	0.75
Type of equipment used	Reverberatory furnace
Stack parameters	
Main stack	
Height	183 m
Diameter	2.6 m
Exhaust gas velocity	28 m/sec
Exhaust gas temperature	135°C
Acid plant	
Height	30.4 m
Diameter	1.8 m
Exhaust gas velocity	16.5 m/sec
Exhaust gas temperature	79°C
Particulate emission rate	
Main stack	247 kg/h
Acid plant	11 kg/h

Zinc Industry Facilities

A zinc smelter produces 99.99+ percent zinc from concentrate containing approximately 62 percent zinc. The zinc concentrates are first roasted at approximately 600°C to convert sulfur to sulfur dioxide and to produce an impure zinc oxide or calcine. The calcine is then transferred to tanks, leached with dilute sulfuric acid, and treated to remove such impurities as lead, gold, and silver.

The leaching step varies somewhat from plant to plant, but the basic process of selective precipitation of the impurities from the leach solution remains the same. This solution is purified and piped to electrolytic cells, where the zinc is electrodeposited on aluminum cathodes. Domestic zinc smelters use electrolytic reduction to reduce the quantity of sulfur and particulate emissions.

The cathodes are lifted from the tanks at intervals and stripped of the zinc, which is melted in a furnace and cast into slabs. Electrolysis of the solution regenerates sulfuric acid that is used in succeeding cycles of leaching.

The parameters of a reference zinc smelter that were used to estimate the radioactive emissions to the air are shown in Table 4.2-10.

Table 4.2-10. Parameters of reference zinc plant (TRI81)

Parameter	Value
Process	Electrolytic reduction
Capacity	88,000 MT/y zinc
Capacity factor	0.8
Radionuclide concentration of input ore	
Uranium-238	0.18 pCi/g
Thorium-232	0.08 pCi/g
Stack parameters	
Number	1
Height	100 m
Diameter	2 m
Exhaust gas velocity	20 m/sec
Exhaust gas temperature	150°C

The reference zinc smelter has a total production capacity of about 88,000 MT of zinc per year, typical of the industry. The plant produces zinc by electrolytic reduction and operates at an annual capacity factor of 0.80, the 1976 industry-wide average (DOI76). Other reference plant parameters are based on actual measurements (EPA82b).

Lead Industry Facilities

Lead smelting involves three distinct processes: sintering, to convert the ore from a sulfide to an oxide or sulfate form and to prepare the feed materials for furnacing; furnacing, to reduce the oxide feed to lead metal; and drossing, to reduce the copper content of the lead bullion from the furnace. After drossing, additional refining steps, as dictated by the specific impurities present and the intended end-use of the product, are performed to yield the purified lead metal.

The parameters of the reference facility that were used to estimate the radioactive emissions to the air are shown in Table 4.2-11 (EPA75, TRI81).

Table 4.2-11. Parameters of reference lead smelter (TRI81)

Parameter	Value
Capacity	220,000 MT/y lead
Capacity factor	0.92
Radionuclide concentration of input ore	
Uranium-238	0.9 pCi/g
Thorium-232	0.5 pCi/g
Stack parameters:	
Number	1
Main stack	
Height	30 m
Diameter	1 m
Exit gas velocity	9 m/sec
Exit gas temperature	90°C
Acid plant stack	
Height	30 m
Diameter	1.8 m
Exit gas velocity	1.7 m/sec
Exit gas temperature	93°C

The reference lead smelter has a capacity of 220,000 MT lead per year, typical of existing plants. The plant operates at a load factor of 0.92, which was the industrywide average for 1979 (DOC80). Other plant parameters are based on a composite of data taken at an operating facility.

4.2.8 Uranium Fuel Cycle Facilities, Uranium Mill Tailings, High-Level Waste Management

Uranium Fuel Cycle Facilities

Uranium fuel cycle facilities are involved in chemical conversion of uranium, isotopic enrichment, fabrication of fuel, and generation of electricity.

Uranium Conversion. Two industrial processes are used for uranium hexafluoride production, the dry hydrofluoride (hydrofluor) method, and the solvent extraction method (EPA73a). The hydrofluor process consists of reduction, hydrofluorination, and fluorination of the ore concentrates to produce crude uranium hexafluoride, followed by fractional distillation to obtain a pure product. The dry hydrofluor process separates impurities either as volatile compounds or as solid constituents of ash. The solvent extraction process employs a wet chemical solvent extraction step at the start of the process to prepare high-purity uranium for the subsequent reduction, hydrofluorination, and fluorination steps. The wet solvent extraction method separates impurities by extracting the uranium into organic solvent and leaving the impurities dissolved in an aqueous solution.

For purposes of estimating radiation doses to the surrounding population, a reference facility was created. This facility utilizes the dry hydrofluor process, which releases radionuclides primarily as gaseous radon, and uranium and thorium compounds in the form of particulates. The parameters of the reference facility are listed in Table 4.2-12.

Table 4.2-12. Parameters of reference uranium conversion facility

Parameter	Value
Type	Fluorination-fractionation (dry hydrofluor) UF_6 plant
Ore grade	Low-impurity plant feed containing 2800 pCi of thorium-230 and 200 pCi of radium-226 per gram of natural uranium
Annual capacity	10,000 MT of uranium
Emission control	Primary treatment, secondary bag filters on dust control streams, and secondary or tertiary scrubbers on process off-gas streams
Stack	
Height	10 m
Plume rise	0.0

Fuel Fabrication. Reactor fuel is fabricated from enriched uranium. The enriched uranium material (uranium hexafluoride) is shipped to fuel producers, who convert it into solid uranium dioxide pellets. These pellets are inserted into zirconium tubes and fabricated into fuel assemblies, which are shipped to the reactors (EPAb78, EPAb73). Emissions consist of uranium, thorium, and protactinium isotopes, which are present as compounds in particulate form.

The parameters of the reference facility developed for assessing the radiological impact of fuel fabrication plants are presented in Table 4.2-13.

Table 4.2-13. Parameters for reference uranium fuel fabrication facility

Parameter	Value
Type of facility	
Ammonium diuranate (ADU)	UF_6 feed to plant hydrolyzed in water, uranium precipitated in ammonia to form ADU. ADU calcined to form UO_2
Direct conversion (DC)	UF_6 feed to plant reacted with water vapor and hydrogen to form UO_2
Capacity	1500 MT/y
Fixed stack height, no plume rise	10 m

Nuclear Power Plants

Nuclear power plants operate on the same general principles as fossil-fuel-fired generating stations. The only significant difference is that a nuclear reactor, rather than a fossil-fuel-fired boiler, supplies the heat to generate steam. The fission process in the reactor produces radioactive gases that may enter the coolant. These contaminants are periodically removed from the coolant and subsequently released in the form of gaseous isotopes such as argon, xenon, and krypton, which are largely inert.

Two basic types of light-water-cooled reactors are currently in use in the United States: boiling-water reactors (BWR) and pressurized-water reactors (PWR). Reference facilities for the two types of commercial reactors, boiling-water and pressurized-water reactors, were developed for the impact analysis of the nuclear power industry (parameters are listed in Table 4.2-14). The reference facilities use a recirculating u-tube steam generator, and their characteristics were developed by the NRC in its environmental statement on light-water-cooled reactors (NRC76, EPA73b).

Table 4.2-14. Parameters for reference light-water reactors

Parameter	Value
Type	Boiling-water reactor and pressurized-water reactor
Capacity	1000 MW(e)
Fuel	Uranium only
Fixed stack height, no plume rise	20 m

Uranium Mill Tailings

As with any ore-processing operation, uranium milling produces large quantities of waste rock. Uranium mill wastes, or tailings, are usually stored in an impoundment located on the mill site. Tailings are usually discharged to the impoundment area as a liquid slurry. Tailings impoundment areas consist of a pond and a dry beach, the sizes of which are based on water recycle rates and evaporation rates. Radionuclide emissions, which are primarily from the dry beach areas, result from wind erosion and diffusion of radioactive gases out of the tailings. The largest radionuclide emission is radon-222 gas.

For purposes of estimating the emissions and health impacts from uranium mill tailings, a reference model was developed and values were assigned to the important parameters (Maa78). These are presented in Table 4.2-15. Because the activity of the mill itself is important for an assessment of the impact of the tailings impoundment, parameters for a model mill are also included in this table (NRC79a, EPA83a, EPA82d).

Table 4.2-15. Parameters for reference uranium mill and tailings impoundment

Parameter	Value
Type of process	Acid-leach solvent extraction
Ore process rate	2000 metric tons per day
Operating days per year	300 days
Mill lifetime	20 years
Ore grade	0.2% U_3O_8
Uranium recovery	95%
Ore activity	560 pCi/g, uranium-238 and daughter products in secular equilibrium
Ore storage area	1 hectare
Ore storage time	10 days
Effective stack height	15 meters
Area of tailings impoundment	60 hectares
Dry beach	15 hectares
Pond and wet beach	45 hectares
Average depth of tailings	12 meters

High-Level Waste Management

In normal operation, uranium fuel-cycle facilities, specifically nuclear reactors and other NRC-licensed facilities, generate high-level radioactive waste, primarily in the form of spent reactor fuels. The option selected for disposal of the spent fuels determines the kind of facilities required for their management. In the interim period, the spent fuels are stored in pools of water, often located at the power-plant or DOE facility.

The reference plant for nuclear generating stations includes releases from spent fuel storage in the form of gaseous krypton and smaller amounts of tritium. For the assessment of uranium fuel-cycling releases, an offsite fuel storage facility was selected as the reference facility. The site parameters (EPA82c) are listed in Table 4.2-16.

Table 4.2-16. Parameters for reference fuel storage facility (EPA82c)

Parameter	Value
Capacity	5,000 tons
Facility life remaining	30 years
Percentage of release respirable	100%
Source type	Point source
Discharge height	100 meters
Distance to site boundary	500 meters

4.2.9 Low-Energy Accelerators

Particle accelerators not operated by DOE are generally low-energy medical and research facilities. The equipment, operational energies, particles accelerated, and target materials used at these facilities vary greatly. Possible sources of radionuclide emissions include loss of target integrity, handling of irradiated targets, and activation of air and dust by the particle beam. The radionuclide emissions are in the form of relatively small quantities of isotopes of oxygen, nitrogen, argon, carbon, and tritium.

Three reference accelerator facilities were developed to assess the health impacts from low-energy accelerators. The parameters assigned to the reference facilities are listed in Table 4.2-17.

Table 4.2-17. Parameters of reference accelerator facilities

Parameter	Value
Type of accelerator	6 MeV Van de Graaff with tritium target, operated 3000 h/y
	18 MeV electron LINAC, operated 2000 h/y
	100 MeV research cyclotron, operated 1000 h/y
Emission control	None
Roof-type stack height	16.8 meters

4.3 Radionuclide Releases

The emission data used in the health impact assessments are summarized in the following subsections. Insofar as possible, measured radionuclide emission data have been used. In the absence of measured data, however, estimates are based on calculated or extrapolated values. The emission data for DOE facilities were obtained from DOE's Effluent Information System for the calendar year 1981 (DOEa81); the data for NRC-licensed facilities were obtained from NRC annual effluent reports; and the data for the other categories, such as coal-fired utility and industrial boilers, uranium and nonuranium mines, and the various extraction industries, were obtained from various reports prepared for the EPA.

More detailed source data for the individual source categories are available in Chapters 2 through 7 in Volume II of this document.

4.3.1 Department of Energy Facilities

The individual DOE facilities were briefly described in the preceding section. Only the largest DOE emission sources are presented in Table 4.3-1 because so many sources are involved. Volume II, Chapter 2, of this document provides detailed emission data for all of the DOE facilities.

Radionuclide emissions from DOE facilities result from three types of operations: (1) nuclear reactor operations, (2) nuclear fuel and weapons materials processing, and (3) accelerator operations. The radionuclide releases resulting from the operation of nuclear reactors are in the gaseous state. The principal radionuclides released are noble gases [argon (AR-41), krypton (Kr-85 and 88), and xenon (Xe-133)] and isotopes of hydrogen (H-2 and H-3). These releases occur during routine purging of radioactive decay products from reactor cooling systems and refueling operations.

Radionuclide releases from nuclear materials processing are primarily particulates, which are released during solid materials handling; however, very small quantities of gaseous radionuclides are released during the processing of spent nuclear reactor fuel elements. The primary particulate emissions from DOE production facilities are uranium (U-234 and U-238). Gaseous releases include tritium and deuterium (H-3 and H-2) and the noble gases listed previously--xenon, argon, and krypton.

Accelerator facilities, the third category of DOE emission sources, release radionuclides in the gaseous state. These emissions result from high-energy particles reacting with air and from the radioactivation of air by secondary particles generated in the accelerator. The primary radionuclides emissions from accelerators are oxygen (O-15), nitrogen (N-13), argon (Ar-41), and carbon (C-11).

4.3.2 NRC-Licensed Facilities and Non-DOE Federal Facilities

As an aid to consistent analysis of NRC-licensed facilities, a reference source was developed for the individual types of facilities included in this category. Radionuclide emission data for the reference NRC-licensed facilities are summarized in Table 4.3-2. Annual radionuclide emission rates are referred to as "source terms" in the computerized models used to estimate health impact.

Table 4.3-1. Summary of radionuclide emissions from DOE facilities

Facility	Radionuclide	Amount released (Ci/y)
Argonne National Laboratory	Ar-41	0.4
	Kr-85	6.7
Brookhaven National Laboratory	H-3	660
	O-15	36,000
	Ar-41	170
Feed Materials Production Center	U-238	0.11
	U-234	0.11
Fermi National Accelerator Laboratory	C-11	1500
Hanford Reservation	H-3	18
	Ar-41	65,000
	Cs-138	11,000
Idaho National Engineering Laboratory	H-3	400
	Ar-41	2,500
	Kr-85	59,000
Lawrence Livermore National Laboratory	H-3	2,600
	N-13	170
	O-15	170
Los Alamos National Laboratory	H-3	1,100
	C-11	130,000
	N-13	25,000
	O-15	200,000
	Ar-41	1,400
	H-3	6,100
Oak Ridge Reservation	H-3	11,000
	Kr-85	6,600
	Xe-133	32,000
Savannah River Plant	H-3	350,000
	Ar-41	62,000
	Kr-85	840,000
	Kr-88	1,500
	Xe-133	3,900

Table 4.3-2. Summary of radionuclide emissions from NRC-licensed facilities and other Federal facilities

Facility	Radionuclide	Amount released (Ci/y)
Research Reactor Reference Facility	Ar-41	8,560
	H-3	22
Accelerator Reference Facility		
Cyclotron	N-13	0.04
	O-15	1.0
	C-11	2.0E-3
Radiopharmaceutical Industry		
Reference Supply Facility	I-125	0.02
	I-131	0.076
	Xe-133	23
	Te-99m	4.5E-3
Reference User Facility	I-125	9.5E-3
	I-125	0.05
	Xe-133	6.4
Reference Sewage Treatment Plant	I-131	5.0E-4
	Te-99m	8.0E-4
Armed Forces Radiobiology Research Institute	Ar-41	1.3
	N-13	3.5E-2
	O-15	3.5E-2
U.S. Army Pulse Reactors	Ar-41	13.3
U.S. Navy		
Reference Nuclear Shipyard	Ar-41	0.41
	C-14	0.10
	Kr-87	0.05
	Xe-135	0.25
Manufacturers of Radiation Sources Reference Facility	H-3	1,060
	Kr-85	61.8
	C-14	4.3
Other NRC Licensees		
Laboratories	H-3	29
Waste Disposal Sites	H-3	6,000
Mineral and Metal Processing Facilities	Rn-222	Not available

4.3.3 Coal-Fired Utility and Industrial Boilers

Both industrial and utility coal-fired boilers emit radionuclides in fly ash. A primary factor influencing the radionuclide content in the fly ash generated during combustion is the type of coal, i.e., its mineral content and the concentrations of uranium, thorium, and their decay products. Other factors affecting radionuclide emissions are the furnace design and capacity, the capacity factor, the heat rate, proportion of fly ash to bottom ash, enrichment factors, and emission control efficiency.

Measurements have shown that trace elements, such as uranium, lead, and polonium, are distributed unequally between bottom ash and fly ash (Be78, Wa82). Although the concentration mechanism is not fully understood, the preferential concentration of certain volatile elements on particle surfaces results in depletion of these elements in the bottom ash and their enrichment in the fly ash (Sm80). The highest concentration of the trace elements in fly ash is found in particulates in the 0.5- to 10.0-micrometer diameter range, the size range that can be inhaled and deposited in the lung. Particulate control devices are less efficient in removing these fine particles than larger particles. Based on measured data, typical enrichment factors are 2 for uranium, 1.5 for radium, 5 for lead and polonium, and 1 for all other radionuclides (EPA81). The radionuclide emissions for the reference utility and industrial boilers are listed in Table 4.3-3. These sources are discussed further in Volume II, Chapter 4, of this document.

Table 4.3-3. Summary of radionuclide emissions from reference coal-fired boilers

Facility	Radionuclide	Emissions (Ci/y)
Utility boiler	U-238	0.1
	Th-230	0.05
	Rn-222	0.96
	Pb-210	0.25
	Po-210	0.25
Industrial boiler	U-238	0.01
	Th-230	0.005
	Rn-222	0.25
	Pb-210	0.025
	Po-210	0.025

4.3.4 Underground Uranium Mines

Radon-222 is the predominant radionuclide released from underground uranium mines. Emissions of uranium and thorium also have been detected at uranium mines, but at levels so low as to be insignificant compared with those of radon. The radon-222, uranium-238, and thorium-232 emissions from the reference underground uranium mine are shown in Table 4.3-4.

Table 4.3-4. Radionuclide emissions from the reference underground uranium mine (EPA83a, Ja80)

Source	Emissions (Ci/y)		
	Radon-222	Uranium-238	Thorium-232
Mine vents	11,000	-	-
Ore, subore, and waste rock piles	500	0.02	3E-4

4.3.5 Phosphate Rock Processing and Wet-Process Fertilizer Plants

The radionuclide stack emissions from the reference fertilizer plant and reference rock drying and grinding plant are listed in Table 4.3-5. More extensive discussions of these facilities and their emissions appear in Volume II, Chapter 6, of this document.

Table 4.3-5. Summary of radionuclide emissions from reference fertilizer plant and reference phosphate rock processing plant

Facility	Radionuclide	Emissions (Ci/y)
Wet-process fertilizer plant (DAP and GTSP combined)	U-238	0.007
	Th-230	0.007
	Pb-210	0.003
	Po-210	0.003
Phosphate rock drying and grinding	U-238	0.015
	Th-230	0.015
	Pb-210	0.015
	Po-210	0.015

4.3.6 Elemental Phosphorus Plants

Polonium-210 and lead-210 are the radionuclides emitted from elemental phosphorus plants in the most significant quantities. More than 95 percent of the polonium-210 and lead-210 are released from the calciner stacks. The high temperature of the calciners volatilizes the polonium-210 and lead-210 from the phosphate rock and results in the release of much greater quantities of these radionuclides than of the uranium, thorium, radium radionuclides. The EPA conducted extensive testing at elemental phosphorus plants, and these data were used to develop the annual radionuclide emission estimates shown in Table 4.3-6.

Table 4.3-6. Estimated annual radionuclide emissions from elemental phosphorus plants

Plant	Emissions (Ci/y)		
	Uranium-238	Lead-210	Polonium-210
FMC[(a)] Pocatello, Idaho	4E-3	0.1	9
Monsanto[(a)] Soda Springs, Idaho	6E-3	5.6	21
Monsanto[(a)] Columbia, Tennessee	2E-3	0.4	0.6
Stauffer[(a)] Silver Bow, Montana	6E-4	0.1	0.7
Stauffer[(b)] Mt. Pleasant, Tennessee	2E-4	0.05	0.1
Occidental[(b)] Columbia, Tennessee	2E-4	0.05	0.1

(a) Based on measured emission rates.

(b) Based on estimated emission rates.

4.3.7 Mineral Extraction Industry

Most of the radionuclide emissions from mineral-extraction facilities are in the form of fine particulates. Lead, copper, zinc, and aluminum facilities were chosen as the reference facilities because each uses high-temperature smelters with the potential for significant releases of particulates.

Radionuclide concentrations in particulates emitted from a smelter are similar to or greater than the concentrations in the materials processed. The radionuclide concentrations greater than those in the original ore are due to the enrichment that takes place when nuclides volatilize during the high-temperature phase of production. Calculations of the releases for the reference smelters are based on the assumption that the radionuclide content in the particulates released is the same as that in the input ore and the application of appropriate enrichment reactors for volatile radionuclides. Multiplying the concentrations of radionuclides in the ore by the total annual particulate release yields the total annual radionuclide release. The radionuclide emissions for the reference facilities in this category are listed in Table 4.3-7. More detailed discussions of the emissions from each facility can be found in Volume II, Chapter 7.

Table 4.3-7. Summary of emissions from reference mineral-extraction facilities

Facility	Radionuclide	Emissions (Ci/y)
Lead smelter	U-238	8.6E-3
	Pb-210	2.6E-2
	Po-210	2.1E-2
Copper smelter	U-238	0.04
	Th-230	2.1E-3
	Pb-210	6.5E-2
	Po-210	0.03
Zinc smelter	U-238	5.6E-4
	Pb-210	2.5E-2
	Po-210	1.5E-3
Aluminum reduction plant	U-238	1.5E-4
	Th-230	2.8E-4
	Pb-210	5.2E-4
	Po-210	4.7E-4

4.3.8 Uranium Fuel Cycle Facilities, Uranium Tailings, High-Level Waste Management

Uranium Conversion Facilities

Conversion facilities handle no irradiated material; therefore, all radionuclides present also occur in nature. These radionuclides are radium, thorium, uranium, and their respective decay products. Uranium is the major source of radioactivity in the gaseous effluents. Possible chemical species of uranium effluents include U_3O_8, UO_2, UF_4, UF_6, and $(NH_4)_2U_2O_7$. In the wet solvent extraction method, uranium is present as uranyl nitrate, which may also appear in gaseous effluents. Thus, the uranium may be released as both soluble and insoluble aerosols. The emissions from the reference facility are listed in Table 4.3-8 (EPA73a, EPA78b).

Table 4.3-8. Atmospheric emissions of radionuclides from the reference uranium conversion facility

Radionuclide	Emissions (Ci/y)
U-238	0.083
Th-234	0.082
Rn-222	9.2

Uranium Fuel Fabrication Facilities

Particulate emissions account for all radionuclides released from fuel fabrication facilities. The radionuclide emissions from the reference fuel fabrication facility are shown in Table 4.3-9.

Table 4.3-9. Radionuclide emissions from the reference fuel fabrication facility (EPA78b, NRC76)

Radionuclide	Emissions (Ci/y)
U-234	0.013
U-235	4.6E-4
U-236	7.0E-4
U-238	1.7E-3
Th-231	4.6E-4
Th-234	1.7E-3
Pa-234	1.7E-3

Light-Water Reactors

Radionuclide emissions from boiling-water reactors (BWR) and pressurized-water reactors (PWR) usually result from contaminants released from the fissionable fuels into the cooling water. These contaminants are removed from the cooling water and periodically released to the atmosphere. A summary of the airborne releases from the reference reactors is presented in Table 4.3-10.

Table 4.3-10. Atmospheric emissions of radionuclides from the reference BWR and PWR facilities (EPA77c)

Radionuclide	Emissions (Ci/y) BWR	Emissions (Ci/y) PWR
I-131	0.2	0.2
Kr-85	300	150
Xe-133	12,000	10,000
H-3	60	400

Uranium Mill Tailings

The amount of airborne emissions from tailings disposal areas depends upon the size of dry tailings beach areas that are subject to wind erosion and radon-222 diffusion. When tailings impoundment areas are almost completely covered by water, radionuclide emissions will be low (NRC79b). The airborne radioactive emissions for the reference uranium mill tailings area due to wind erosion and gaseous diffusion are listed in Table 4.3-11.

Table 4.3-11. Radionuclide emissions from the reference uranium mill (EPA83b)

Radionuclide	Emissions (Ci/y)(a)
U-238	8.9E-3
U-234	8.9E-3
Th-230	1.2E-1
Ra-226	1.2E-1
Pb-210	1.2E-1
Po-210	1.2E-1
Rn-222	4.4E+3

(a) During the operational phase of the mill.

High-Level Waste Management

Airborne emissions from the reference fuel storage facility result from venting the cask gases and from activity in the cask unloading and fuel storage pools. The radionuclide emissions for the reference fuel storage facility are shown in Table 4.3-12.

Table 4.3-12. Radionuclide emissions from reference storage facility (EPA82c)

Radionuclide	Emissions (Ci/y)
H-3	2.4
Kr-85	890

4.3.9 Low-Energy Accelerators

Emissions of radioactive materials at accelerator facilities are produced by two principal mechanisms: 1) the activation of air by accelerated particles or secondary radiation, which results in radioactive carbon, nitrogen, oxygen, or argon; and 2) the loss of radioactive material (usually tritium) from a target into the air. Airborne radionuclide releases from the three reference accelerator facilities are presented in Table 4.3-13.

Table 4.3-13. Radionuclide releases from reference low-energy accelerators (EPA79c) (Ci/y)

	Type of accelerator		
Radionuclide	100 MeV cyclotron	18 MeV electron linac	6 MeV Van de Graaff
C-11	2.0E-3	--	--
N-13	0.04	--	--
O-15	1.0	--	--
H-3	--	--	1.0
C-14	--	1.0E-9	--
Ar-41	--	1.0E-4	--

4.4 Uncertainties

Quantifying radionuclide emissions from the source categories addressed in this report necessitated the review and summarization of significant amounts of data collected by numerous agencies. The emission data presented for facilities in this chapter were gathered from a variety of information sources. These information sources include reports prepared by individual facilities, tests conducted for standards development not associated with radionuclide emissions, engineering determinations of expected releases from physical and chemical processes, and tests conducted specifically for determination of radionuclide emissions.

It was neither practical nor feasible to evaluate every single facility for individual contributions to health risk; therefore, for certain source categories, a single reference facility that best characterized the industry was either selected or developed from existing source emission data. The use of a single facility to represent a large number of sources simplifies the assessment of health risks, but the potential for errors increases because of the necessary assumption that all relevant factors used in the analysis of the reference facility are in fact representative of the industry.

In those cases where measured emission data were not available for a facility or process, an assessment of the expected releases was based on an engineering analysis of the process generating the release. These mass and chemical balances require assumptions regarding process parameters. As with the selection of reference facilities, airborne emissions determined through the use of mass balances may include an expected level of uncertainty due to the required assumptions. Similarly, some annual radionuclide emission rates are based partially on fugitive particulate emission factors. The fundamental nature of fugitive emissions makes them extremely difficult to quantify precisely, and emission factors represent the mean estimate of emissions, which can vary substantially due to wind, humidity, material handling practices, and other factors.

Even annual radionuclide emission rates based on physical and radiological measurements are not exact. Any physical measurement is subject to uncertainties imposed by the accuracy and precision of the sampling methodology and analytical procedures used. Of these two factors, imprecision in sampling (and sample handling/preparation) generally presents the greater uncertainty. Determination of the radioactivity of a sample is fairly straightforward; the significant uncertainties result from the random and systematic errors of an instrument or method. Analytical problems can occur when several different radionuclides are collected in one sample and must be determined individually. Considering the uncertainty in both sampling and analysis, emission measurements for radionuclides are generally accepted as being accurate within approximately ±20 percent at best. In general, the range of uncertainty in annual radionuclide emission rates based on physical and radiological measurements are expected to be comparable.

Other factors that can increase the overall uncertainty of the emission data are as follows:

(1) The use of enrichment and partitioning factors that were determined from a single source for a particular radionuclide.

(2) The use of data not specifically collected to quantify radionuclide emissions.

(3) The adequacy of quality control and quality assurance procedures followed during the collection and analysis of samples.

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Chapter 5: REDUCTION OF DOSE AND RISK

5.1 Introduction

Genetic and somatic health effects due to radionuclide emissions can be limited by two basic strategies: (1) the application of emission control technology, and (2) the implementation of work practice requirements. These two control strategies are documented in this chapter. The particular facilities for which each is applicable are identified, and the factors that create uncertainties in the evaluation of the efficiency of these and other procedures in the reduction of radionuclide emissions are described.

5.1.1 Emission Control Technology

Emission control technology implies the installation of a piece of equipment that removes radionuclides from flue gas prior to its discharge to the air. The most widely used emission control devices are scrubbers, filters, charcoal adsorbers, cyclonic collectors, and electrostatic precipitators (ESPs). These and other less common control devices are discussed in Section 5.2. Some of these devices are unique and have only limited application, but all have been demonstrated to be effective in reducing radionuclide emissions.

5.1.2 Work Practices

Work practice procedures are techniques that reduce radionuclide emissions at the source by process modifications or refinements. Work practices include procedures that reduce radionuclide emissions by reducing the radionuclide content of the process, and processes that minimize the amount of radionuclides entering the flue gases.

Fugitive emissions are emissions that escape from roof monitors, doors, storage piles, exposed soil surfaces, etc., rather than from a stack or vent. If necessary, fugitive emissions are usually reduced through the implementation of specific work practices. Examples include applying earth covers, wetting arid areas, and enclosing conveying equipment. Brief descriptions of the various types of fugitive emission control are presented in this chapter; more detailed information can be found in the references.

5.1.3 Impact of Existing Regulations on Strategies for Reducing Emissions

Gaseous and particulate emissions from several of the source categories discussed in this report are currently regulated by existing Federal and state standards. A brief discussion of emission standards is pertinent to this chapter because many of the existing techniques for emission reductions have been developed and refined in response to these standards.

Particulate emissions from coal-fired boilers with a heat input greater than 250 million Btu per hour are regulated under Section 111 of the Clean Air Act (36 FR 24878, December 23, 1971). The Subpart D New Source Performance Standard (NSPS) requires coal-fired boilers constructed after August 17, 1971, be equipped with control equipment that limits particulate emissions to 43 ng/J (0.1 lb/million Btu heat input). Emissions from new utility boilers (construction commencing after September 18, 1978) are also regulated under Section 111 of the Clean Air Act (44 FR 33580, June 11, 1979). The Subpart Da NSPS limits particulate emissions to 13 ng/J (0.03 lb/million Btu heat input) and requires previously uncontrolled sulfur dioxide (SO_2) emissions to be reduced by up to 90 percent. The EPA recently proposed (49 FR 25102, June 19, 1984) a Subpart Db NSPS that will limit particulate emissions from new industrial coal-fired boilers with a heat input of between 100 and 250 million Btu per hour to 22 ng/J (0.05 lb/million Btu heat input). The emission limitations imposed by these three NSPS (Subparts D, Da, and Db) require the installation and operation of best available control technology (BACT). Electrostatic precipitators or fabric filter systems are usually installed to meet the particulate emission standards, and state-of-the-art flue gas desulfurization (FGD) systems are installed to achieve the required SO_2 reductions.

Emissions from the phosphate industry are also controlled by existing Federal and state regulations. Particulate emissions from new or modified phosphate rock drying and grinding facilities are regulated by the Subpart NN NSPS (47 FR 16582, April 16, 1982). These standards are usually met by installing ESPs and high-energy scrubbers on dryers and fabric filters on grinders. Phosphate fertilizer plants use wet-scrubber systems to reduce fluoride emissions. These controls, which are also effective in reducing particulate emissions, are necessary to comply with the emission limits imposed by Subparts T through X (40 FR 33152, August 6, 1975).

A more detailed discussion of the regulation of radionuclides for NRC, DOE, and other facilities was presented in Chapter 2.

5.2 Emission Control Technology

Radionuclides from most source categories are released to the air as particulate matter; only a few, such as isotopes of radon, iodine, and noble gases, are released in the gaseous state. Therefore, control of particulate emissions is the most effective means of reducing radionuclide emissions for most source categories.

The key parameter for evaluating the effectiveness of a control technology is its collection efficiency. The efficiency of a control device is the ratio of the amount of pollutant removed to the amount of pollutant entering. Particulate control efficiency can be expressed in terms of weight, particle number, or radioactivity of pollutant removed; however, unless stated otherwise, collection efficiency is assumed to be based on the weight. If the weight is measured over the entire particle size range or distribution, the efficiency is referred to as the overall collection efficiency. Collection efficiency can be computed for one or more particle size ranges, however, and when this is done, efficiency is reported as fractional collection efficiency.

Penetration is another term that is sometimes used in describing the performance of a control device. Penetration is the ratio of the amount of pollutant passing through the control device to the amount of pollutant entering the device. The sum of penetration plus efficiency for a control device must equal 1.

Several additional considerations merit discussion in the context of evaluating the effectiveness of an emission control technology in reducing radionuclide dose and risk. If a process involves high temperatures (e.g., a combustion process), some radionuclides can be volatilized during the process. As the flue gas cools before its discharge to the atmosphere, some of the radionuclides may condense on the surface of nonradioactive particulate matter (i.e., the nonradioactive particles function as condensation nuclei). Such condensation normally takes place preferentially on particles with a high surface-to-volume ratio. This phenomenon results in an increase in the concentration of condensed volatile radionuclides on smaller-sized particulate emissions. This is generally referred to as fine particle enrichment. Nevertheless, particulate matter with condensed radionuclides behave the same as other particles and can be collected by regular particulate control equipment.

The focus of the remainder of this section is on descriptions of various control devices available to reduce radionuclide emissions and identification of the facilities where these control devices can be used. Because most of these control devices were not designed specifically to remove radionuclides, explanations emphasize the operating principles by which the devices collect nonradioactive particulate matter and gases.

5.2.1 Scrubbers

Scrubbers can be installed on a variety of process exhaust streams and can serve numerous functions. For example, in phosphate fertilizer processes, scrubbers can serve economical purposes by recovering and conserving ammonia (NH_3). Scrubbers also efficiently reduce gaseous and particulate emissions. For the latter purpose, scrubbers are currently used on coal-fired boilers and in phosphate, elemental phosphorus, and mineral extraction industries. They are also used by NRC facilities (PNL83).

Despite the many designs and applications, the fundamental process of all scrubbers is the same. In each case, the gas and liquid phase streams are mixed, and the gaseous and/or particulate components of the gas stream are absorbed and removed from the process by the liquid stream. The process for disposal of the waste stream can be either "wet" or "dry," depending on the liquid-recovery design. Spray-tower, packed-bed, tray-tower, venturi, and wet centrifugal scrubbers are examples of the types of scrubbers that are in commercial use.

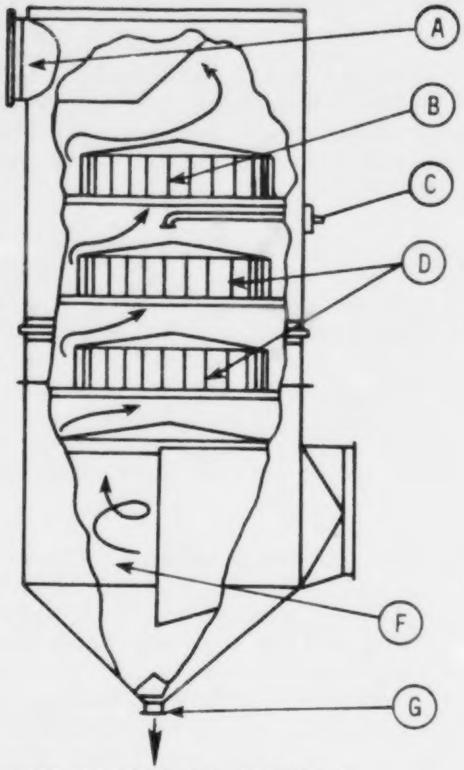
For reduction of radionuclide emissions, most scrubbers function as a particulate control device and often constitute only part of an overall control system that may also include filters, scrubbers, mist eliminators, charcoal adsorbers, and other devices (TRI79). Figure 5.2-1 illustrates two designs that have proven to be effective in particulate control. These and other wet scrubbers reduce radionuclide emissions from sewage treatment plants, light-water-reactor fuel-fabrication facilities, uranium conversion plants, separation and waste calcining facilities, uranium "yellowcake" processing and packaging, and elemental phosphorus plants. (Yellowcake is the final precipitate formed in the milling of uranium, consisting of various forms of triuranium octoxide, U_3O_8). For example, a high-energy venturi scrubber applied to the exhaust of one elemental phosphorus calciner provided about 97 percent removal efficiency for polonium-210 (DM80).

Scrubbers are most effective in removing larger particulate matter (greater than 1 micrometer in diameter) and can be more practical than filters for exhaust streams with high moisture content. Typical scrubber applications are exhausts from ore dryers and sewage treatment plants (PNL83). Depending on particle size in the exhaust and the type of scrubber, efficiencies can range from about 93 percent for a baffle-type scrubber to 99+ percent for a high-energy venturi scrubber (DM80). As previously discussed, some radionuclide sources are regulated by EPA's New Source Performance Standards. Two such sources, coal-fired boilers and the phosphate fertilizer industry, must operate scrubbers to achieve sulfur dioxide and fluoride emission reductions, respectively, and these scrubbers also reduce radionuclide emissions.

5.2.2 Filters

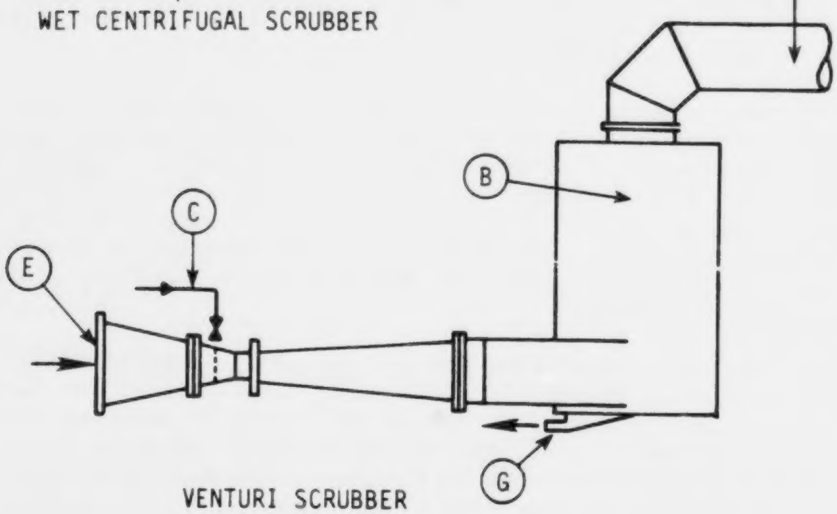
Filters are one of the most frequently used radionuclide emission reduction devices. Various designs provide effective particulate control in each of the nine source categories except underground uranium mines. The effluent characteristics (e.g., volume, temperature, and type of particulate) of the source categories may differ greatly, but most filter designs can accommodate a wide range of operating conditions. Filters are extremely versatile and can be used to supplement other control equipment, such as ESPs and mechanical collectors.

The types of filters used to reduce radionuclide emissions include high-efficiency particulate air (HEPA), fabric, sintered-metal, and sand filters. Efficiencies of HEPA filters, as reported by vendors, are



SYMBOLS	PARTS
A	CLEAN-AIR OUTLET
B	ENTRAINMENT SEPARATOR
C	WATER INLET
D	IMPINGEMENT PLATES
E	DIRTY-AIR INLET
F	WET CYCLONE FOR COLLECTING HEAVY MATERIAL
G	WATER AND SLUDGE DRAIN

WET CENTRIFUGAL SCRUBBER



VENTURI SCRUBBER

Figure 5.2-1. Wet scrubber particulate control devices (PNL83).

99.97 percent (DM80). Efficiencies of sand filters are about 99 percent (DM80). Except for the sintered metal type, filters have been used in many different applications.

Sintered-metal filters, shown in Figure 5.2-2, are used in fluoridation (uranium conversion from UF_4 to UF_6) processes. Sintered-metal filters also have been tested at pilot-plant fluidized-bed calciner and spray calciner facilities and have achieved 99.999 and 99.9 percent removal efficiencies, respectively (PNL83).

Fabric filters (baghouses), shown in Figure 5.2-3, are utilized primarily in uranium conversion, coal-fired boilers, and phosphate and other mineral-extraction industries (TRI79). Fabric filters provide a medium-cost, high-efficiency particulate-removal system. A properly designed and maintained fabric filter generally achieves a removal efficiency of 99 percent or greater, depending on the application. Depending on the design and material of construction, filter surfaces are cleaned by vibration or by reversing the direction of the gas flow (PNL83). This allows continual use of fabric filters with relatively little maintenance.

For many years, sand filters similar to those shown in Figure 5.2-4 have been installed as particulate and radionuclide control devices at DOE facilities. Such filters are ideal for high-temperature, large-volume, exhaust streams. Sand filters have high removal efficiencies equal to those of a single-staged HEPA filter. Maintenance costs are low, but a large area is required for installation and operation. Examples of sand filter applications include uranium and plutonium recovery and separation and solid waste processing facilities (TRI79). Some uncertainty surrounds the use of sand filters, however, because of the lack of disposal standards for spent filters (PNL83).

HEPA filters (Figure 5.2-5) may be the most applicable to the nine source categories addressed in this report. These filters can control particulate emissions effectively with a median removal efficiency of 99.97 percent for 0.3-micrometer particles. The pressure drop across the system is approximately 1 in. H_2O, assuming gas flow rates are within the designed range. The HEPA filters can function as single-stage or multistage filters, and they are frequently installed along with other control devices.

Each filter has certain inherent limitations. Sintered-metal filters are limited by their relatively small range of past and current applications. Fabric filters, which are restricted by moisture content, must operate in medium to lower temperatures, which prohibits their use on high-temperature exhausts such as those normally associated with calciners. In addition to requiring large areas, sand filters have the drawbacks of high capital costs and large pressure drops across the systems. A similar filter system that uses a fiberglass filter medium instead of sand reduces the required area and pressure drop, but the removal efficiency is usually lower. Although not necessarily a limitation, the disposal nature of HEPA filters is an important consideration.

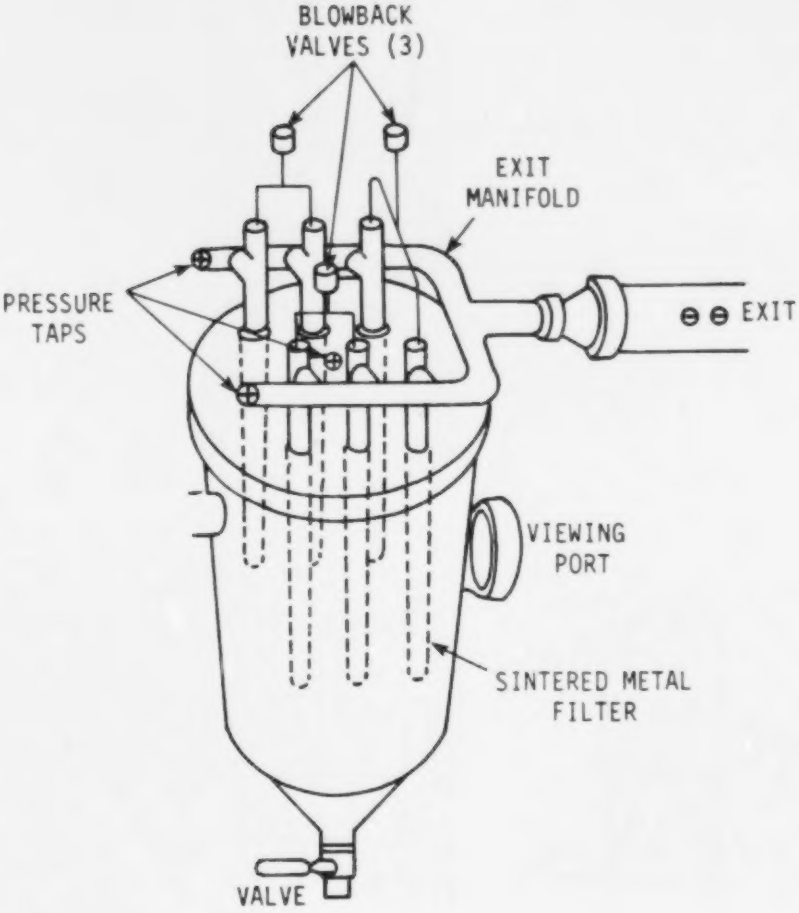

BLOWBACK VALVES (3)
EXIT MANIFOLD
PRESSURE TAPS
⊖ ⊖ EXIT
VIEWING PORT
SINTERED METAL FILTER
VALVE

Figure 5.2-2. Pilot-plant sintered-metal filter.

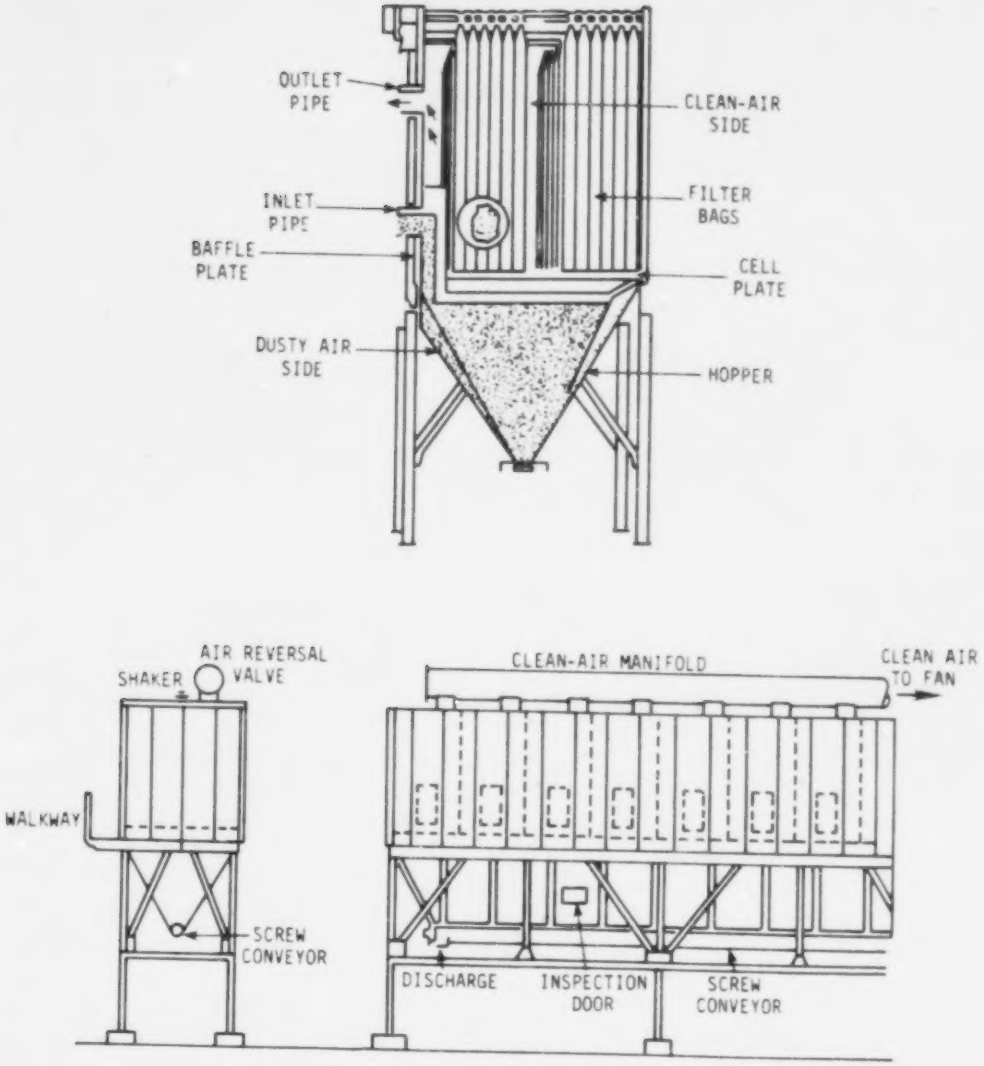
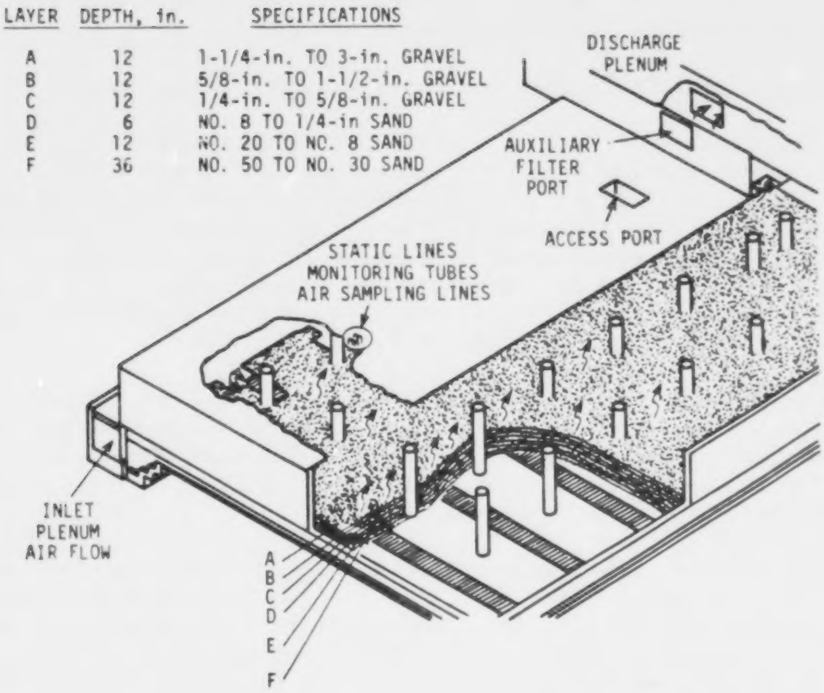

OUTLET PIPE
CLEAN-AIR SIDE
INLET PIPE
FILTER BAGS
BAFFLE PLATE
CELL PLATE
DUSTY AIR SIDE
HOPPER
AIR REVERSAL VALVE
SHAKER
CLEAN-AIR MANIFOLD
CLEAN AIR TO FAN
WALKWAY
SCREW CONVEYOR
DISCHARGE
INSPECTION DOOR
SCREW CONVEYOR

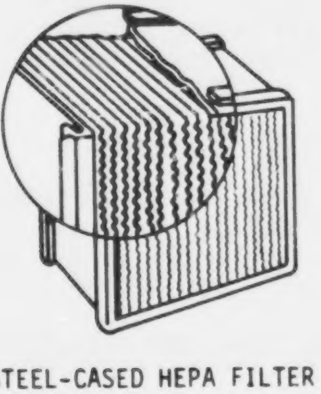
Figure 5.2-3. Fabric filters.



LAYER	DEPTH, in.	SPECIFICATIONS
A	12	1-1/4-in. TO 3-in. GRAVEL
B	12	5/8-in. TO 1-1/2-in. GRAVEL
C	12	1/4-in. TO 5/8-in. GRAVEL
D	6	NO. 8 TO 1/4-in SAND
E	12	NO. 20 TO NO. 8 SAND
F	36	NO. 50 TO NO. 30 SAND

DISCHARGE PLENUM
AUXILIARY FILTER PORT
ACCESS PORT
STATIC LINES
MONITORING TUBES
AIR SAMPLING LINES
INLET PLENUM AIR FLOW
A
B
C
D
E
F

Figure 5.2-4. Multilayered sand filter.



STEEL-CASED HEPA FILTER

Figure 5.2-5. Open-face HEPA filter.

Also, the use of HEPA filters is limited to ambient air exhaust stream conditions, which may require that moisture separators or other control devices be installed upstream of these filters.

5.2.3 Mechanical Collectors and Electrostatic Precipitators

Mechanical collectors, illustrated in Figure 5.2-6, separate particles from the gas stream by centrifugal and gravitational forces. The collection efficiency of a mechanical collector is a function of configuration and particle size. For a double-vortex cyclone, reported separation efficiencies exceed 99 percent for particles greater than 6 micrometers and 95 percent for particles greater than 1 micrometer (Ae). Generally, mechanical collectors are used only as precleaners upstream of an electrostatic precipitator or a fabric filter.

Electrostatic precipitators (ESPs), shown in Figure 5.2-7, use high-voltage sources to charge the particles in the gas stream, which are then collected on large metal plates. The collecting plates are periodically cleaned by rapping. The efficiencies of ESPs can exceed 99.9 percent, depending on the application.

Electrostatic precipitators and mechanical collectors are currently used to reduce radionuclide emissions from coal-fired boilers and from mineral-extraction facilities. Mechanical collectors are also used at separation and waste calcining facilities and at sewage plants. Phosphate fertilizer and milling industries generally use filters, but ESPs or mechanical collectors may be substituted (TRI79). Likewise, both devices can be used to supplement filter controls for other specific processes.

The process limitations of mechanical collectors and ESPs are minimal. These devices are usually designed to handle large variations in exhaust temperature, volumetric flow rates, and particulate loading. They may not perform well when handling saturated effluents, but the most restrictive performance limitation for either device is its ability to collect submicron particulate matter. If an exhaust stream is characterized by fine particles, filters are generally the more efficient (PNL83, DM80).

5.2.4 Charcoal Adsorbers

All the previously discussed control devices are used primarily to reduce particulate radionuclide emissions. Gaseous radionuclide emissions are controlled by charcoal adsorbers and other devices, which are described in this and subsequent subsections.

Charcoal adsorbers (sometimes referred to as charcoal filters or carbon filters) are applied throughout the source categories addressed in this report. These include waste management facilities, storage vaults, power generating and research reactors, hot cells, weapons test facilities, plutonium production, and radiation-source manufacturing facilities (TRI79). Charcoal filters (adsorbers) are not used in place

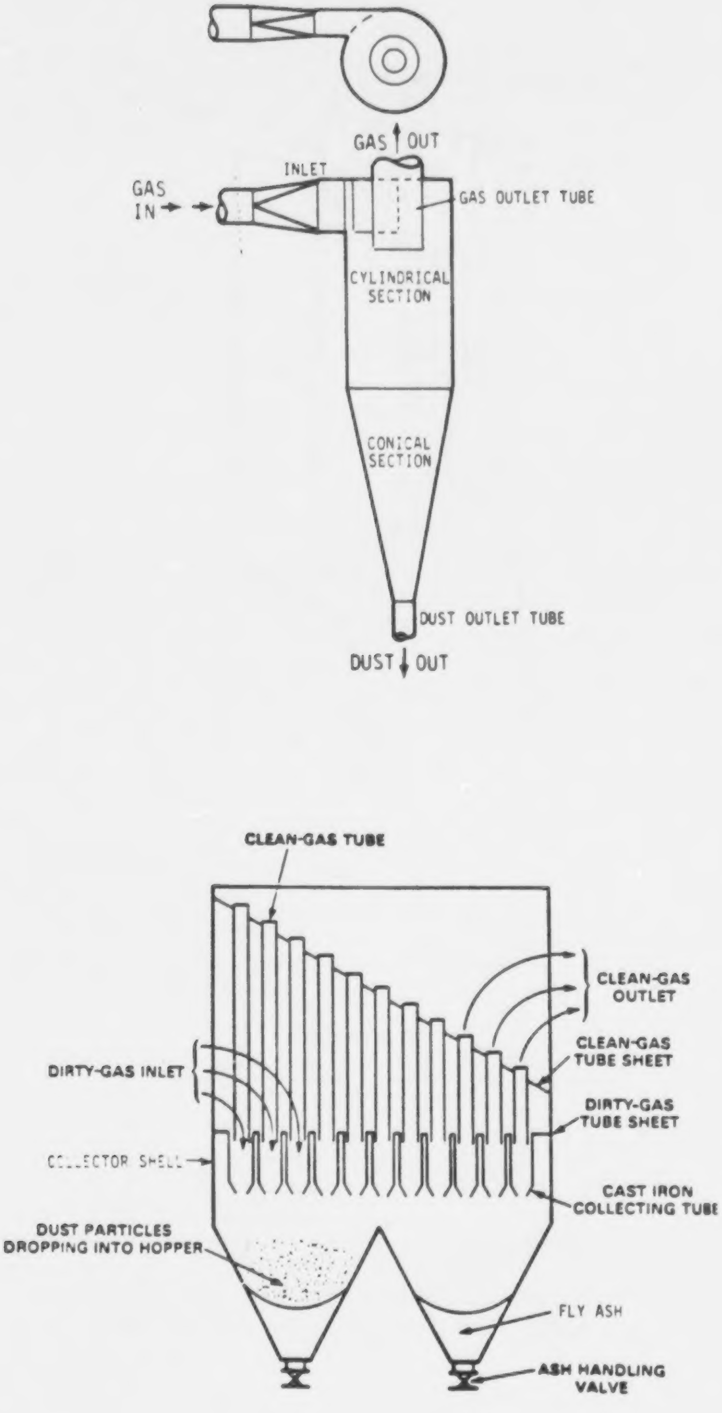
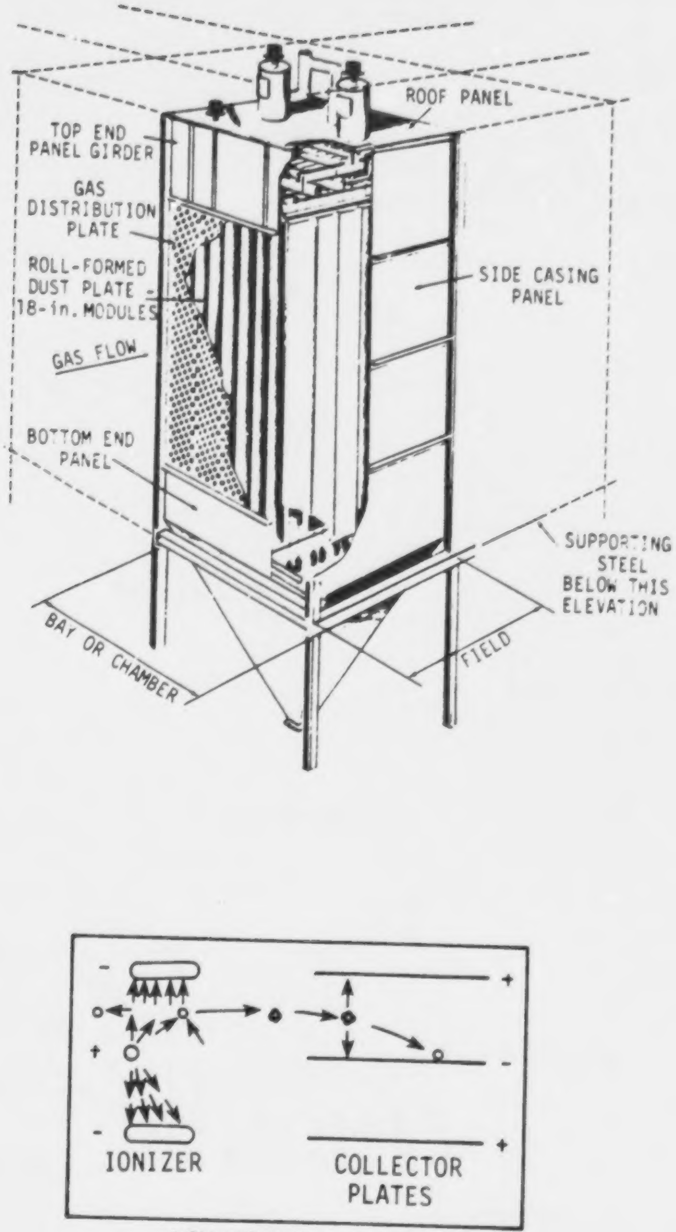

GAS OUT
INLET
GAS IN
GAS OUTLET TUBE
CYLINDRICAL SECTION
CONICAL SECTION
DUST OUTLET TUBE
DUST OUT
CLEAN-GAS TUBE
CLEAN-GAS OUTLET
CLEAN-GAS TUBE SHEET
DIRTY-GAS INLET
DIRTY-GAS TUBE SHEET
COLLECTOR SHELL
CAST IRON COLLECTING TUBE
DUST PARTICLES DROPPING INTO HOPPER
FLY ASH
ASH HANDLING VALVE

Figure 5.2-6. Mechanical collectors.


ROOF PANEL
TOP END PANEL GIRDER
GAS DISTRIBUTION PLATE
ROLL-FORMED DUST PLATE - 18-in. MODULES
SIDE CASING PANEL
GAS FLOW
BOTTOM END PANEL
SUPPORTING STEEL BELOW THIS ELEVATION
BAY OR CHAMBER
FIELD
IONIZER
COLLECTOR PLATES

LOW-VOLTAGE DESIGN
12,000 TO 13,000 VOLTS ON IONIZER
6,000 TO 7,000 VOLTS ON COLLECTING PLATES

Figure 5.2-7. Electrostatic precipitators.

of particulate filters. In fact, they are often installed in series with HEPA filters or other particulate control devices.

In most instances, charcoal adsorbers are used to reduce radioiodine emissions; however, these adsorbers can be used, along with chilled traps, to collect and store krypton and xenon gases. Because these gases have relatively short half-lives, radionuclide dose and risk can be reduced by preventing the gases from being released until their radioactivity has decreased. Manufacturers of radiation sources utilize this method. Charcoal adsorbers can also be used to remove antimony from hot-cell exhausts (PNL83).

Despite attractive (possibly 99.9 percent) radioiodine removal efficiencies, some important limitations must be considered before charcoal adsorbers are installed. Flow rate, humidity, temperature, iodine concentration, adsorber bed age, and other parameters affect removal efficiencies and may reduce them to 90 percent. For example, as temperature increases, iodine desorbs from charcoal. Also, in certain effluent streams, charcoal may ignite at temperatures as low as 180°C, and the presence of nitrogen oxide-nitrogen dioxide ($NO-NO_2$) may cause spontaneous ignition (PNL83).

5.2.5 Miscellaneous Emission Control Equipment

A few unique control technologies are used to control radionuclides. These include silver-based sorbent systems, oxidation/adsorption processes, cryogenic distillation, and purge cascades, which -- although not widely used -- have been shown to be effective in reducing gaseous and particulate radionuclides.

Silver-Based Sorbent Systems

Silver-based sorbent systems can employ solid or liquid sorption techniques. Solid sorbent systems use reactant pellets in a pellet bed; liquid sorbent systems use a concentrated scrubber solution in a packed tower. Despite the process differences, the emission control capabilities of both systems are similar.

In general, both solid and liquid sorbent systems have proven to be effective radioiodine control devices for certain processes. They are currently used at waste-management and fuel-processing facilities. Silver-based sorbent systems operate most effectively in low-flow exhaust streams or in the removal of trace amounts of iodine downstream of other control devices. With few exceptions, solid and liquid sorbent systems are chemically and thermally stable and therefore function efficiently when charcoal adsorbers might be unsafe or ineffective. Despite their similarities in application, the particular process mechanisms and parameters of solid and liquid sorbent systems should be discussed independently.

Most solid sorbent control devices are similar in design and operating parameters. Solid sorbent control devices use silver zeolite or silver nitrate silica pellets in pellet beds that are 5 to 20 cm thick.

Operating temperatures should be higher than 110°C to prevent moisture interference. Regeneration of pellet beds has not been perfected; therefore, the use of two pellet beds is recommended so that the spent reagents in one pellet bed can be replaced while the other bed is operated. Limitations of these sorbent systems are the instability of some silver zeolite reagents when used on acidic exhausts from reprocessing facilities, lack of knowledge regarding the chemistry of the sorption processes, and uncertainties regarding decreased efficiencies caused by organic vapors, other halogens, and sulfur compounds.

The only liquid sorbent control system in existence is one in which iodine is removed by coating Berl saddles or other types of ceramic packing with a concentrated silver nitrate solution. Exhaust gases pass over the coated packing at about 190°C, and radioiodine is collected as silver iodide or silver iodate on the ceramic packing. As silver nitrate is consumed, the packing is recharged by flushing with a fresh silver nitrate solution. Proper operation of this system requires accurate temperature control and frequent packing regeneration. As with solid sorbents, liquid sorbent iodine capacities are reduced in the presence of other halogens (PNL83).

Oxidation/Adsorption Processes

The oxidation/adsorption process is a potential means of controlling tritium emissions from various reactors and processing facilities. At low concentrations, tritium must be converted to tritiated water to be effectively trapped. Thus, the process for removing tritium from exhaust gases involves two steps: (1) oxidation of tritium to tritiated water, and (2) removal of tritiated water. Oxidation of tritium is accomplished by the use of catalysts or metal oxides. For example, tritium from a process with an operating temperature of about 177°C can be effectively oxidized with platinum or palladium catalysts. Similar results can be obtained with a metal oxide, such as copper oxide, on processes operating at temperatures between 500° and 700°C. In both cases, hydrogen can be added to the exhaust stream to enhance tritium oxidation. Furthermore, the metal oxide beds can be regenerated with air at temperatures between 300° and 500°C.

The absorption of tritiated water involves cooling the exhaust, passing it through a condenser, and then through a desiccant. The exhaust cooler reduces the gas temperature to approximately 21°C (room temperature). The condenser removes as much of the tritiated water as possible before the gas stream enters the desiccator. This reduces the dewatering required by the desiccator and also the frequency at which new desiccant is needed or desiccant regeneration is required. The desiccator uses silica gel, molecular sieve, or other desiccants to remove moisture still entrained in the gas stream.

Overall removal efficiencies vary with design criteria and operating parameters (such as bed depth, flow rate, and temperature). Tritium emissions can be reduced below normal detection levels (0.1 to 1.0 $\mu Ci/m^3$) of an ionization counter; however, the numerous operating conditions involved make it impossible to state specific reduction efficiencies (PNL83, DM80). Tritium removal efficiencies as high as 99.9999 percent have been reported (PNL83).

Cryogenic Distillation

For several years, rare gases have been processed commercially by cryogenic distillation. This same cryogenic process can be used to reduce radioactive noble gas emissions from sources such as fuel reprocessing plants and reactors. Krypton and xenon are the most common noble gases removed by cryogenic distillation. After removal, these condensed gases are stored until their radioactivity has decreased to a level that is safe for release.

The distillation process is a complex multistage system. Exhaust gases must be pretreated before they enter the cryogenic distillery. Potentially hazardous or troublesome gases, such as carbon dioxide, nitrogen oxides, and oxygen, must be removed from the exhaust stream. After pretreatment, the exhaust stream enters a two-stage separation process. In the first stage, krypton and xenon are separated from the other gases by the use of liquid nitrogen in a countercurrent stripping column. In the second stage, a fractioning column is used to separate the krypton and xenon for storage (PNL83).

Purge Cascades

Purge cascades are a series of traps through which an exhaust stream must pass before being vented to the atmosphere. Depending on the filtering media being used, these traps can remove a wide range of radionuclides. Purge cascade traps may contain particulate filtering media or scrubbing media. Media such as sodium fluoride and alumina are used to control particulate emissions related to uranium and thorium processes. Caustic scrubbing solutions, such as sodium hydroxide or potassium hydroxide, lower the radioiodine concentrations in exhaust streams and also reduce particulate emissions. In addition, some traps are refrigerated to increase their radionuclide reduction efficiency. These cascades are used at several gaseous diffusion plants to reduce gaseous and particulate emissions (DM80). Efficiencies range from 85 to 100 percent, depending on design, application, and filter media used (DM80).

5.3 Work Practices

Work practices are process modifications, refinements, or techniques that reduce radionuclide emissions at their source. Some work practices were developed to improve process performance and have incidentally proven to be effective in reducing radionuclide emissions and dose and risk. Many of these practices are currently being implemented.

Delayed venting involves the use of storage drums, holding tanks, or retention bags to delay venting of the exhaust so that radioactive gases with short half-lives can partially or totally decay before being emitted to the atmosphere (DM80). Unlike traps or adsorbers, delayed venting neither physically nor chemically alters the exhaust, but merely allows time for radionuclide decay. Delayed venting techniques can be applied to power generating reactors, radiopharmaceutical facilities, and accelerator facilities.

Evacuation of accelerator tubing is a work practice applied to accelerators. The air in the accelerator tubes is evacuated to reduce the amount of air made radioactive during operation and thus the radioactive emissions. When evacuations are impractical, pure inert gases may be used to fill voids where air would normally contact activated surfaces. Research and test reactor operators implement this technique to reduce radioactive noble gas emissions, primarily argon.

Work practice techniques in underground uranium mining include the use of mine wall sealants, bulkheading, mine pressurization, and backfilling. Sealants have been able to reduce radon emissions in underground uranium mines by 56 percent. Although sealants have not yet been proven to be effective because of their high cost, research continues for the development of better sealants. Bulkheading, as shown in Figure 5.3-1, involves sealing off mined-out stopes. This practice can reduce mine-air radon concentrations up to 60 percent. Pressurizing a mine, as shown in Figure 5.3-2, retards radon diffusion into mine air; however, its effect on surface emissions has been estimated to be about 20 percent. Backfilling entails refilling mined-out areas with waste or dirt; this procedure can control up to 80 percent of the radon emissions.

Several types of work practices, including washing ore and wet grinding, are applicable to uranium and phosphate processing. Washing process feed rock to reduce its initial dust concentration before the milling process has proven to be effective in controlling particulate emissions. The residence time of the ore in dryers or calciners, or the extent to which an ore is dryed or calcined, determines the amount of fine dust in the product and, consequently, the amount of particulate emissions. Wet-grinding systems, a viable alternative to dry-grinding processes, emit fewer particulates and eliminate the need to dry the feedstock (TRI79). Wet-grinding phosphate systems are currently used to reduce particulate emissions at two facilities.

Mining and milling industries use work practices to reduce fugitive emissions. The control of fugitive emissions from uranium, phosphate, and other metal and nonmetal mining/milling processes primarily reduces particulates and radon gas (Ko80). These controls include earth covers, wetting of arid areas, and covered transport facilities.

Earth covers which consist of layered soil approximately 3 meters deep are frequently used on waste piles, reclaimed lands, or inactive surface mining areas to reduce both particulate and radon emissions.

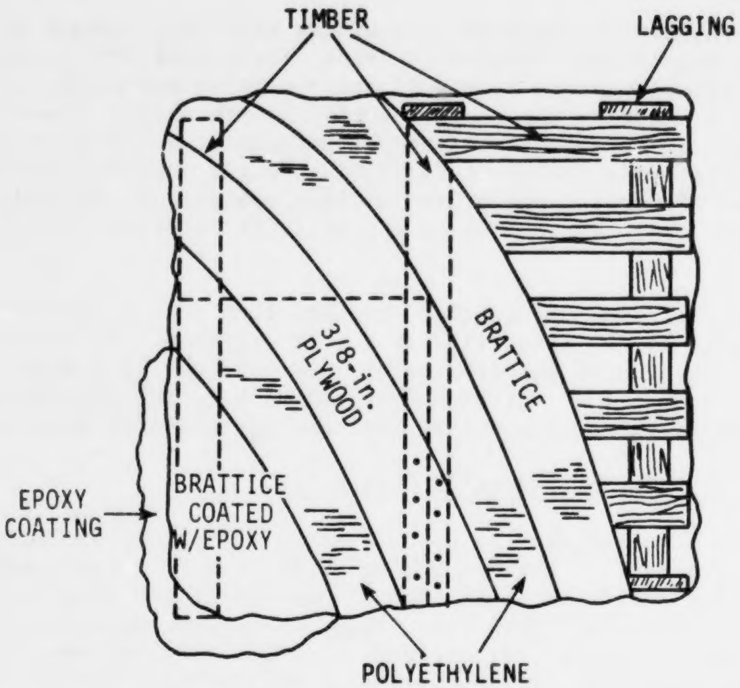

TIMBER
LAGGING
3/8-in. PLYWOOD
BRATTICE
EPOXY COATING
BRATTICE COATED W/EPOXY
POLYETHYLENE

Figure 5.3-1. Bulkheading of mine stopes.

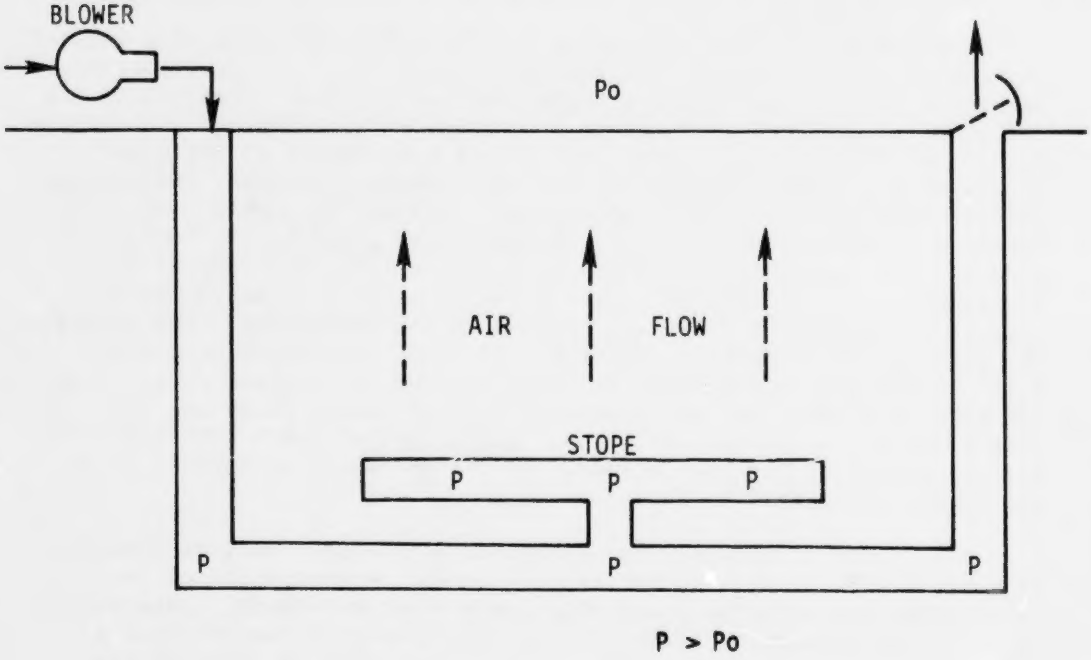

BLOWER
Po
AIR
FLOW
STOPE
P
P
P
P
P
P
P > Po

Figure 5.3-2. Mine pressurization.

Earth covers may not be practical for mining areas and storage piles that are only temporarily inactive because of the need for frequent access. Fugitive emissions from arid storage piles and mining areas can be controlled by wetting the exposed surfaces with water. Chemical sprays (as opposed to water) are used occasionally, but only to coat waste piles. Covering transport facilities (e.g., conveyor systems), which are used throughout the mining/milling operations, not only reduces emissions, but also conserves and protects resources (TRI79, DM80).

Controlled land use is another strategy that reduces population exposure, but it is not classified as a work practice. By owning and controlling the use of a buffer area of land surrounding a mine, mining companies can reduce the radiation dose and risk to the population without necessarily reducing actual radionuclide emission rates.

5.4 Summary of Emission Reduction Strategies

Table 5.4-1 summarizes the major control technology applications. Although certain control devices may be applicable to a particular source, a multitude of processes within that source may require independent control devices; therefore, Table 5.4-1 also includes supplemental controls. Because fugitive and process techniques have limited or no supplemental controls, however, most of the supplemental controls shown are source control devices.

5.5 Uncertainties in Evaluation of Control Technology Efficiencies

A key parameter for evaluating the overall performance of a control technology is its removal or collection efficiency. A more important parameter in terms of reducing radiation dose and risk, however, is its radionuclide collection efficiency. Collection efficiencies are usually determined in one of two ways: (1) direct measurement of pollutant levels (e.g., stack testing), or (2) mass/material balance. Efficiency calculations based on direct measurement require the simultaneous measurement of the radioactivity of the pollutant entering the control device and the radioactivity of the pollutant exiting the control device. Efficiency calculations that use a material balance do not directly measure the amount of pollutant emitted to the atmosphere. For example, the particulate collection efficiency of an ESP installed on a coal-fired boiler can be estimated by measuring the ash content of the coal, the coal feed rate, and the amount of fly ash collected by the ESP. In this example, the difference between the weight of ash entering the ESP and the weight of fly ash collected by the ESP would be assumed to be the amount discharged to the air.

Additional uncertainty is associated with quantifying radionuclide collection efficiency. In the above example, determination of particulate collection efficiency was relatively straightforward. Determining radionuclide collection efficiency is complicated by factors such as fine particle enrichment and the physical and chemical forms of the radionuclides.

Table 5.4-1. Summary of emission reduction strategies

	Control technologies				Work practices	
Source category and/or affected facility	Scrubber	Filters	Mechanical collectors and ESPs	Charcoal adsorbers	Process techniques	Fugitive emission controls
Research and test reactors		P		I	NG	
Hot cells		P		I,Sb		
Commercial waste management		P		I		P
Plutonium fuel fabrication		P				
Plutonium production reactor		P		I		
Radiopharmaceutical	P	P	P	I,NG	NG	
Separation/waste calcining	P	P	P	I		
Elemental phosphorus	P	P				P
High-level waste tank farm		P				
Extraction industry	P	P	P		P	P
Plutonium glovebox/storage vault		P				
Phosphate industry	P	P	P		P	P
Accelerators		P			NG	
Mining						Rn
Coal-fired boilers	P	P	P			
Uranium conversion	P	P				
Light-water reactor fuel fabrication	P	P				
Power generating reactors		P		I,NG	NG	
Uranium milling	P	P	P			P,Rn
Weapons test sites		P		I	NG	

Legend for types of radionuclide gases removed:

P = Particulates--uranium, plutonium, and others
I = Iodine
NG = Noble gases--argon, krypton, and xenon
Rn = Radon
Sb = Antimony

If the actual percentage of fine particle enrichment is unknown or is known to fluctuate with process changes, the use of particulate collection efficiency to estimate radionuclide collection efficiency adds still another degree of uncertainty. For example, a particulate control device may be known to have an overall collection efficiency of 99 percent; however, if a process is characterized by significant enrichment, the radionuclide collection efficiency may be considerably less than 99 percent because of the higher concentration of radionuclides on the fine particles that are in the 1 percent fraction that is not removed by the control device. Also, a high-temperature process can volatilize radionuclides (e.g., Po-210) that are otherwise in the solid (particulate) state. If a process is equipped with a particulate control device and some fraction of radionuclides is volatilized, the radionuclide collection efficiency is uncertain and becomes dependent on quantifying the amount of volatilization that has occurred.

All physical measurements required to calculate efficiency are subject to uncertainties imposed by the precision and accuracy of the sampling methodologies and the analytical procedures. Sampling uncertainties not only include variabilities in the procedures used to collect the sample (e.g., repeatability and reproducibility of the method), but also such variabilities as the representativeness of the sample collected and the representativeness of process operation conditions at the time of sampling. Thus, the uncertainty associated with sample collection is difficult to quantify. On the other hand, quantifying the uncertainty associated with analytical procedures is more straightforward and can be accomplished by computing a 95 percent confidence interval for each analysis.

Despite the uncertainties involved in determining control technology performance, control efficiencies usually do not vary dramatically. For example, a high-energy scrubber with a specified efficiency of 99 percent will normally operate within a percentage point of this value as long as the equipment is operated in accordance with design specifications. Furthermore, when the uncertainty of all the elements in the overall radionuclide risk assessment process is considered, the uncertainty associated with quantifying control technology performance does not appear to be a major contributor to the overall uncertainty in the final assessment results.

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DM80 Dames and Moore, Airborne Radioactive Emission Control Technology, unpublished report prepared under EPA Contract No. 68-01-4992, White Plains, New York, 1980.

Ko80 Kown B. T., et al., Technical Assessment of Radon-222 Control, Technology for Underground Uranium Mines, Bechtel National, Inc., Report prepared under EPA Contract No. 68-02-2616, Task 9, 1980.

PNL83 Pacific Northwest Laboratory, Control Technology for Radioactive Emissions to the Atmosphere at U.S. Department of Energy Facilities (Draft), PNL-4621, March 1983.

TRI79 Teknekron Research, Inc., Technical Support for the Evaluation and Control of Emissions of Radioactive Materials to Ambient Air, McLean, Virginia, 1979.

Chapter 6: MOVEMENT OF RADIONUCLIDES THROUGH ENVIRONMENTAL PATHWAYS

6.1 Introduction*

This chapter describes how airborne radionuclides are transported in the environment from the point of release into the air up to the final concentration in various compartments of the environment, where these radionuclides can affect human beings. The objective of Chapter 6 is to describe the environmental pathways that are considered at the U.S. Environmental Protection Agency in evaluating radionuclide concentrations in air, soil, and food that result from airborne releases of radioactivity from various facilities. In this context, facilities are not only those normally associated by the public with radioactivity, such as national laboratories and uranium processing plants, but also other mineral processing plants, fossil fuel combustion facilities, etc.

The airborne environmental pathways are shown in Figure 6.1-1. Starting the process, the radionuclide sources release the materials in the form of particulates or gases, forming a plume that disperses downwind (Section 6.2). Concentrations of these radionuclides in the air can directly affect people in two ways: through external dose caused by photon exposure from the plume, or through internal dose resulting from radionuclide inhalation. As the airborne radionuclides move from the point of release, they (especially those in particulate form) deposit on ground surfaces and vegetation as a result of dry deposition and precipitation scavenging (Section 6.3). Photon radiation from the radionuclides deposited on the ground also contributes to the external doses. Finally, small fractions of the radionuclides deposited on plant surfaces and agricultural land enter the food chains, concentrating in produce and in animal products such as milk and meat (Section 6.4). Consumption of contaminated foodstuff then contributes to the internal doses of radiation to individuals.

Radionuclide concentrations in air, on soil surfaces, and in food products can be calculated by using the computer code AIRDOS-EPA. A description of the code and some examples of its applications, with an overview of the uncertainties associated with its predictions, appear in Section 6.5.

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*Technical terms such as radioactivity, exposure, dose, and photon radiation are defined in Chapter 7.

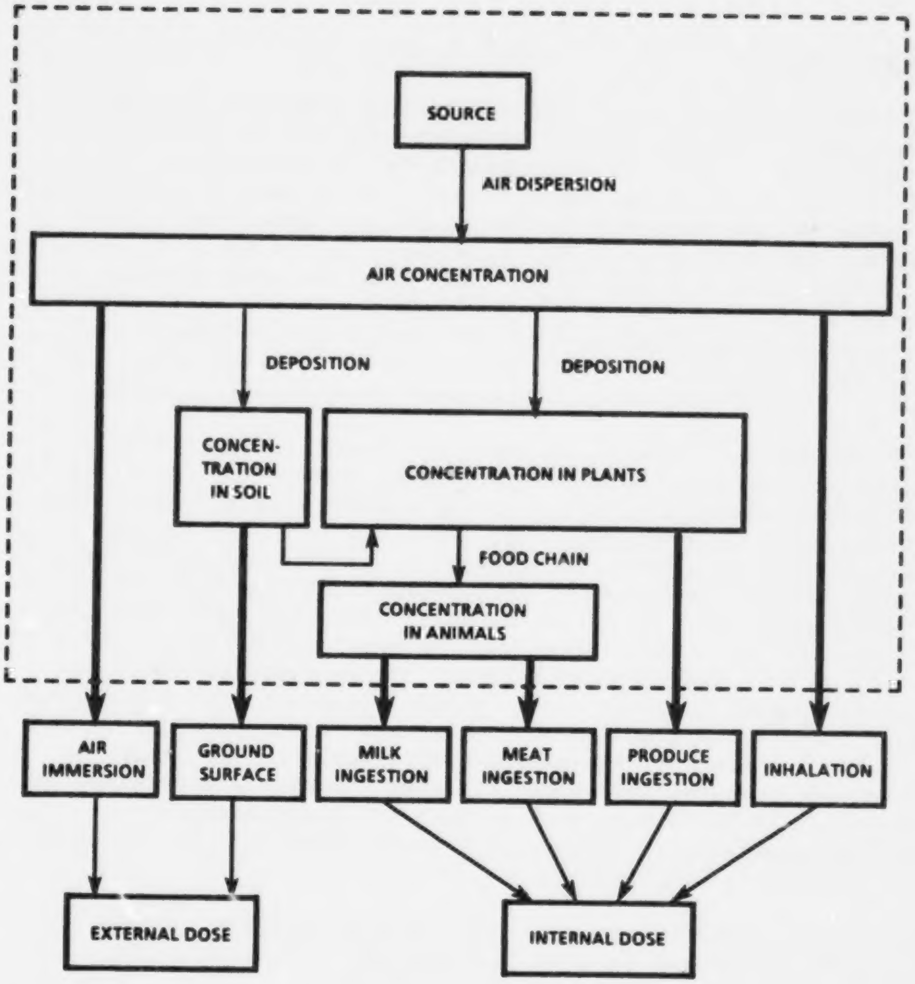
SOURCE
AIR DISPERSION
AIR CONCENTRATION
DEPOSITION
DEPOSITION
CONCEN-TRATION IN SOIL
CONCENTRATION IN PLANTS
FOOD CHAIN
CONCENTRATION IN ANIMALS
AIR IMMERSION
GROUND SURFACE
MILK INGESTION
MEAT INGESTION
PRODUCE INGESTION
INHALATION
EXTERNAL DOSE
INTERNAL DOSE

Figure 6.1-1. Pathways of airborne radionuclides into the environment.

This chapter gives an overview of the basic environmental processes considered by EPA in assessing atmospheric releases of radionuclides. See references Ha82, Ti83, and NCRP84 for a more detailed description of the processes, modeling techniques, and uncertainty estimates.

6.2 Dispersion of Radionuclides Through the Air

6.2.1 Introduction

Radionuclides entering the atmosphere are transported away from their point of release and are diluted by atmospheric processes. To perform a radiological assessment, it is necessary to model the long-term average dispersion resulting from these processes. This is because the sources under consideration release radionuclides at rates that are substantially uniform when considered over long periods of time, and because the somatic and genetic effects on human health are generally treated as being the result of chronic exposure over long periods of time.

As large-scale winds move over the earth's surface, a turbulent boundary layer, or mixed layer, is created that controls the dispersion of the released radionuclides. The depth and dispersion properties of the mixed layer, which are highly variable over short periods of time, are controlled by two sources of turbulent effects: mechanical drag of the ground surface and heat transfer into or from the boundary layer. The mechanical drag of the ground surface on the atmosphere creates a shear zone that can produce significant mechanical mixing. The mechanical mixing is stronger when the wind is stronger and the roughness elements (water, grains of dirt, grass, crops, shrubs and trees, buildings, etc.) are larger. The vertical scale (dimension or thickness) of the mechanical mixing zone is related to the size of these roughness elements. Heat transfer into or from the boundary layer, the second source of turbulent effects, also strongly affects the mixed layer's turbulent structure and thickness. Solar heating creates huge rising bubbles or thermals near the ground. These large bubbles produce turbulent eddies of a much larger scale than those from the mechanical drag of the ground surface. With strong solar heating on a clear day, the mixing layer may be a few thousand meters deep. On a clear, calm night the boundary layer virtually disappears, so that radionuclides (and other pollutants) are dispersed with very little turbulent diffusion.

The objective of the atmospheric transport models used by EPA is to incorporate the essential physical data necessary to characterize an extremely complex turbulent flow process into a simplified model that is adequate to predict the long-term dispersion of radionuclide releases. In general, the data necessary to implement a detailed theoretical model of atmospheric dispersion are not available and would be impractical to obtain. Apart from the data problem, the mathematical complexities and difficulties of a direct solution to the turbulent dispersion problem are profound and beyond the practical scope of routine EPA regulatory assessments. The widely accepted alternative has been to incorporate experimental observations into a semi-empirical model such as outlined below that is practicable to implement.

Three basic meteorological quantities govern dispersion: wind direction, wind speed, and stability. Wind direction determines which way a plume will be carried by the wind: a wind from the northwest moves the plume toward the southeast. Although wind direction is a continous variable, wind directions are commonly divided into 16 sectors, each centered on one of the cardinal compass directions (e.g., north, north-northeast, northeast, etc.). Since there are 16 sectors, each one covers a 22-1/2-degree angle. Wind speed directly influences the dilution of radionuclides in the atmosphere. If other properties are equal, concentration is inversely proportional to wind speed. This raises the question of what happens in a calm. A wind too light to turn an anemometer (about 0.5 m/s) and therefore recorded as a calm can still disperse an atmospheric release. Customary wind speed categories include 0 to 3 knots* (lowest speed) to greater than 21 knots (highest speed).

Atmospheric stability, the third meteorological quantity, categorizes the behavior of a parcel of air when it is adiabatically (without heat transfer) displaced in a vertical direction. If the displaced parcel would be expected to return toward its original position, the category is stable; if it would continue to move away from its original position, the category is unstable. Under conditions of neutral stability, the parcel would be expected to remain at its new elevation without moving toward or away from its old one. Typically, the conditions associated with the unstable classes are very little cloud cover, low wind speeds, and a sun high in the sky. The atmosphere is neutral on a windy, cloudy day or night, and is stable at the surface at night when the sky is clear and wind speeds are low. Dilution due to vertical mixing occurs more rapidly with increasing distance under unstable conditions than under stable ones. Stability categories range from A (very unstable) to D (neutral) to G (very stable).

A table of frequencies (fractions of time) for each combination of stability, wind direction, and wind speed is the starting point for any assessment of long-term atmospheric dispersion.

6.2.2 Air Dispersion Models

EPA uses a Gaussian model for most radionuclide dispersion calculations. The model also includes consideration of such processes as plume rise, depletion due to deposition, and radionuclide ingrowth and decay.

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*A knot is one nautical mile per hour. A nautical mile is 1852 meters.

Gaussian Plume Model

The basic workhorse of EPA dispersion calculations is the Gaussian model. Hanna et al. (Ha82) have listed several reasons why the Gaussian model is one of the most commonly used. These are quoted below:

"(1) It produces results that agree with experimental data as well as any model.

"(2) It is fairly easy to perform mathematical operations on this equation.

"(3) It is appealing conceptually.

"(4) It is consistent with the random nature of turbulence.

"(5) It is a solution to the Fickian diffusion equation for constants K and u.

"(6) Other so-called theoretical formulas contain large amounts of empiricism in their final stages.

"(7) As a result of the above, it has found its way into most government guidebooks, thus acquiring a 'blessed' (sic) status."

The long-term Gaussian plume model gets its name from the shape presumed for the vertical concentration distribution. For a ground level source, the concentration is maximum at ground level and decreases with elevation like half of a normal or Gaussian distribution. For an elevated release, the concentration is symmetrically distributed about the effective height of the plume, characteristic of a full Gaussian distribution. Actually the vertical dispersion is limited by the ground surface below and any inversion lid* above the release (see Fig. 6.1-2). At large distances from the point of the release, the concentration becomes uniformly distributed between the ground and the lid. Within each of the 16 direction sectors, the concentration is considered to be uniform at any given distance from the release. For a ground-level release, the ground-level concentration decreases monotonically with distance from the release point; for an elevated release, the ground-level concentration increases, reaches a maximum value, and then decreases with increasing distance from the release point.

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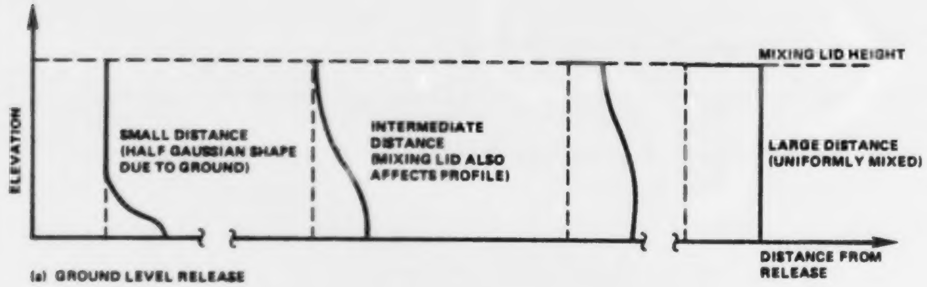
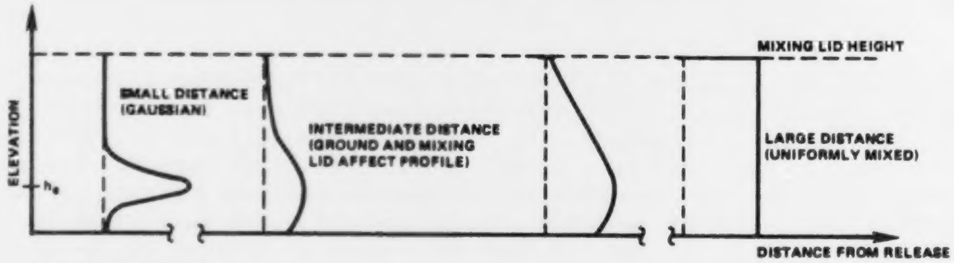
*An inversion lid is defined by the altitude in the atmosphere where the potential temperature begins to increase with increasing height, thus limiting the volume of air available for diluting releases.


MIXING LID HEIGHT
ELEVATION
SMALL DISTANCE (HALF GAUSSIAN SHAPE DUE TO GROUND)
INTERMEDIATE DISTANCE (MIXING LID ALSO AFFECTS PROFILE)
LARGE DISTANCE (UNIFORMLY MIXED)
DISTANCE FROM RELEASE

(a) GROUND LEVEL RELEASE


MIXING LID HEIGHT
ELEVATION
h_e
SMALL DISTANCE (GAUSSIAN)
INTERMEDIATE DISTANCE (GROUND AND MIXING LID AFFECT PROFILE)
LARGE DISTANCE (UNIFORMLY MIXED)
DISTANCE FROM RELEASE

(b) ELEVATED RELEASE

Figure 6.1-2. Vertical concentration profiles for plume versus downwind distance from release.

Mathematically, the long-term average dispersion calculation used by EPA can be expressed as

$$\chi/Q = \frac{2.03 \exp[-0.5(h_e/\sigma_z)^2]}{u \times \sigma_z} \tag{6-1}$$

where χ/Q (s/m^3) is the concentration for a unit release rate at a distance x(m) from the release point, h_e(m) is the effective height of the release, σ_z(m) is the vertical dispersion parameter appropriate to the stability category and distance x, and u(m/s) is the wind speed. At distances where the release is uniformly mixed between the ground and lid, the expression becomes

$$\chi/Q = \frac{2.55}{u \times h_\ell} \tag{6-2}$$

where h_ℓ(m) is the lid height and the other quantities are the same as before.

Plume Rise Model

Vertical momentum or buoyancy can cause a plume to rise to an effective height that is several times the physical height of the release. The momentum flux of a release is proportional to the product of the volume flow rate and the vertical exit velocity while the buoyancy flux is proportional to the product of the volume flow rate and the difference between the temperatures of the release gases and the ambient air. Momentum rise is initially dominant for most plumes, even though buoyant rise may become the more important process at larger distances. In any case, plume rise increases with distance from the release point; the effective height of the plume may not reach a limiting value until the plume is several kilometers from the point of release.

Plume Depletion Model

As radionuclides in the plume are dispersed, their activity is depleted by dry deposition and precipitation scavenging.

The rate of plume depletion due to dry deposition and precipitation scavenging is proportional to the deposition rate (see 6.3). ORP uses a source depletion model which considers the shape of the vertical concentration profile to be unchanged by depletion. Depletion due to deposition generally does not cause more than half of the released activity to be removed at a distance of 80 km. Depletion by precipitation scavenging occurs only during periods of precipitation.

Radiological Decay and Ingrowth

Radiological decay can also reduce the concentration in the plume. A typical elapsed time for traverse between the point of release and a receptor located 80 km away is about 5 hours. Thus, only nuclides with short half-lives would be appreciably depleted by radiological decay. For example, argon-41, which has a 1.8 hour half-life, decays to about 15 percent of its original activity in 5 hours.

When a released radionuclide is a parent for other radionuclides in a chain, those decay products will become part of the plume's activity even though they were not released by the source. For example, cesium-137 is the parent of barium-137m, which has a half-life of about 2.6 minutes. The barium-137m activity would reach 90 percent of that of the cesium-137 in about 8.5 minutes, the time required at a typical wind speed of 5 m/s for the release to travel about 2.5 km. For many nuclides, the radiological effects associated with exposure to decay products are at least as important as those from exposure to the parent. For example, the external photon dose from a release of cesium-137 is entirely due to photons from its decay product barium-137m.

6.2.3 Uncertainties in Atmospheric Dispersion Modeling

EPA must deal with several uncertainties in its modeling of atmospheric dispersion. Two basic considerations contribute to these uncertainties. The first involves the parameters that enter into the model and how well they are known or can be determined for a particular situation. The presumption is that the basic assumptions for which the model was developed are satisfied and that the uncertainty of predicted concentrations depends primarily on the uncertainty of the data used in the calculations. The second consideration involves the use of a modeling technique under conditions that do not satisfy the basic assumptions for which the model was developed. Such use may be the only practicable alternative available for assessing atmospheric dispersion, but the principle uncertanties are now related to evaluating the significance of these effects that are not considered in the model. An example of this would be the use of the Gaussian plume model, which was developed for short distances over an open, flat terrain, to assess dispersion over large distances or in a complex terrain dominated by hills and valleys.

In regard to the first consideration, the authors of NCRP84 concluded that the determination of appropriate basic parameters such as wind speed and direction can be accomplished so that they are not major contributions to model uncertainty. However, the uncertainties associated with derived parameters (such as stability class) or lumped parameters (such as those used to characterize deposition, resuspension, or building wake effects) can dominate the model uncertainties.

The effect of the uncertainty of an input variable can strongly or weakly influence the model output depending upon circumstances. For example, the effective height of a release, h_e, can be estimated using a plume rise model to within a factor of about 1.4 (NCRP84). From equations (6-1) and (6-2), it is clear that when σ_z is much smaller than h_e that the effect of this uncertainty on equation (6-1) is strong; whereas at large distances where equation (6-2) is appropriate, the value of h_e has little effect on the calculated concentration at all.

Little and Miller (Li79 and Mi82) have surveyed a number of validation studies of atmospheric dispersion models. Although these studies provide limited data, they indicate an uncertainty of approximately a factor of 2 for annual average concentrations for locations within 10 km of the release and approximately a factor of 4 (77 percent of their samples) to 10 (92 percent of their samples) for locations between 30 and 140 km of the release. The validation studies were for fairly complex terrain, i.e., substantial hills and valleys but not extreme conditions of either terrain or meteorology.

6.3 Deposition of Atmospheric Radionuclides

6.3.1 Introduction

Atmospheric deposition includes a complex set of processes that result in the transfer of radionuclides from the plume to the ground surface and vegetation. Processes are categorized as dry when they result in the direct transfer from the plume to the surfaces in contact with it and wet when the transfer is first from the plume to precipitation and then from the precipitation to the ground or vegetation surfaces.

6.3.2 Dry Deposition Model

Dry deposition models generally relate the surface deposition flux to the air concentration at some reference height, typically 1 meter above the ground. The resulting equation is

$$W = v_d \, X_o \qquad (6\text{-}3)$$

where W is the deposition flux to the surface (Ci/m^2s), X_o is the reference height air concentration (Ci/m^3), and v_d is the deposition velocity (m/s). Although v_d has the units of a velocity (hence its name), it is a lumped variable relating the deposition flux to the air concentration. The value of the deposition velocity depends on a complex interaction of effects--atmospheric, aerosol, and surface (canopy). Thus while the deposition velocity is often assigned a simple fixed value, it actually represents the result of a diverse combination of effects.

6.3.3 Wet Deposition Model

Wet deposition models relate the flux due to precipitation scavenging to the concentration in the plume. Since the activity scavenged from the plume by an element of precipitation is presumed to remain with the precipitation element until reaching the ground surface, the deposition flux is proportional to the total wetted activity in a vertical segment of the plume (Ci/m^2). The resulting equation can be expressed as

$$W = \lambda_{sc} \bar{X} L \tag{6-4}$$

where W is the surface flux (Ci/m^{2}s), $\bar{X}$ is the average wetted air concentration (Ci/m^3), L is the depth of the wetted layer (m), and λ_{sc} is the scavenging rate (s^{-1}). λ_{sc} is a variable that lumps together the complex interactions between precipitation and the plume. Because the deposition flux is proportional to the vertically integrated concentration (i.e., the total activity in a column of unit ground surface area), it is independent of the effective height of the release. Raising the effective height of a release lowers the dry deposition flux but leaves the flux resulting from precipitation scavenging unchanged.

6.3.4 Soil Concentration Model

The deposited radionuclides accumulate in the surface soil until they are removed either by radiological decay or by processes such as leaching. The areal concentration can be expressed as

$$C_a = W \frac{1-\exp(-\lambda_B t_b)}{\lambda_B} \tag{6-5}$$

where C_a is the areal concentration (Ci/m^2), W is the radionuclide flux to the ground surface (Ci/m^{2}s), t_b(s) is the time for radionuclide buildup in soils, and λ_B is the effective removal rate from soil (s^{-1}). When the deposited radionuclide is the parent of other radionuclides, their soil concentrations at time t_b due to ingrowth from the parent must also be calculated.

For calculating root transfer to crops, the radionculide concentration in the surface soil layer can be expressed as

$$C_s = C_a/P \tag{6-6}$$

where C_s is the soil concentration (Ci/kg) and P is the areal density of dry soil (kg/m^2) for the plowed or mixed soil layer.

The value of t_b, the deposition accumulation time, is typically in the range of 20 to 100 years. For nearby individual assessments, t_b is chosen to correspond to the expected operational life time of the facility. If EPA considers it likely that the facility would be replaced by another similar one at that time, then t_b is increased accordingly up to a maximum value of 100 years. Of course, only those environmental concentrations which depend on soil deposition are affected by the choice of t_b. For collective (population) assessments, a value of 100 years is used for t_b. This value corresponds to establishing a 100-year cutoff for the time following a release when any significant intake or external exposure associated with deposition on soil might take place. Since radionuclide inhalation is generally the dominant risk pathway, total risk is not sensitive to the choice of t_b.

The value of λ_B is the sum of the radiological decay constant, λ, and an environmental removal rate for deposited radionuclides from soil, λ_s. Hoffman and Baes (Hob79) considered a simplified leaching-loss model appropriate to agricultural soil for calculating λ_s. Their range of values for the parameter K_D (the equilibrium distribution coefficient relating the ratio of the radionuclide concentration in soil water to that on soil particles) for Cs is from 36.5 to 30,000 ml/g. The corresponding ratio of λ_s is 820:1. The uncertainty in λ_s is also significantly affected by the uncertainty in the other parameters as well. Although their model is a reasonable one, adequate studies for its validation do not exist. Since the choice of appropriate values for λ_s is so uncertain, EPA has used 0.2 y^{-1} as a general nominal value (the geometric mean of λ_s for Pu^+, I^-, Cs^+, and Sr^{2+} ions is 1.2 10^{-2} y^{-1} using Hoffman and Baes median data values) and a value of 0.1 y^{-1} for urban settings where strong surface runoff would be expected to increase the effective removal rate.

6.3.5 Uncertainties

Uncertainties in v_d and λ_{sc} are substantial; NCRP84 lists measured values of v_d which vary over three orders of magnitude. Hanna et al. (Ha82) note that "The use of scavenging coefficient for wet removal modeling is probably best regarded as an order of magnitude estimation procedure". Actually much of the wide range of values reflects measurement uncertainties as well as actual variations. Furthermore most field deposition measurements reflect short-term or episodic studies rather than long-term observations. Miller and Little (Mi82) concluded that the data necessary to quantify the accuracy of calculated ground concentrations are not currently available.

6.4 Transport through the Food Chain

6.4.1 Introduction

Deposited radionuclides may become associated with vegetation by two principal routes: (1) direct interception of a fraction of the deposited activity by plant surfaces, and (2) transfer of deposited activity from the soil through the plant's root system. Radionuclides in animal feed crops such as pasture grass or stored feeds can be transferred to foods such as milk and meat.

6.4.2 Concentration in Vegetation

The plant concentration due to interception of the deposition flux can be calculated as (Baa76)

$$C_v^d = W \frac{f_r}{Y_v} \frac{T_v \; (1-\exp(-\lambda_E t_e))}{\lambda_E} \tag{6-7}$$

where C_v^d is the crop concentration (Ci/kg) at harvest, W is the deposition flux (Ci/m^2s), f_r is the fraction of the deposition flux which the vegetation intercepts, Y_v is the vegetation yield (kg/m^2), T_v is a translocation factor, λ_E is the effective removal rate of the intercepted radionuclide from the vegetation (s^{-1}), and t_e is the exposure time of the vegetation to the radionuclide flux (s). Miller (Mi79) has observed that data for f_r and Y_v are well represented by the expression

$$f_r = 1-\exp(-\gamma Y_v) \tag{6-8}$$

where γ was found to range between 2.3 and 3.3 m^2/kg when Y_v is expressed in kg/m^2, dry. Since the product γY_v is generally less than 1.0, for many practical purposes (6-8) can be approximated as

$$f_r = \gamma Y_v \tag{6-9}$$

In this case the quantity f_r/Y_v (6-7) can be replaced by γ which shows much less environmental variation than f_r and Y_v do separately. Note that Y_v is the total vegetative yield which can be several times the edible portion yield for a crop. T_v, the translocation factor, relates the radionuclide concentration in the edible portion to that in the entire plant. Baker et al. (Baa76) suggest a value of 1.0 for leafy vegetables and fresh forage, and 0.1 for all other produce. (A value of 1.0 is used for all crops in AIRDOS-EPA.)

The value for λ_E is the sum of λ, the radionuclide decay constant and λ_w, the weathering rate factor. For a typical weathering half-life of 14 days, λ_w has a value of $5.7 \; 10^{-7} \; s^{-1}$. In general, the product $\lambda_E \; t_e > 1$ and (6-9) can be simplified to

$$C_v^d = W \frac{f_r \; T_v}{Y_v \; \lambda_E} \tag{6-10}$$

Radionuclides also transfer directly from the soil to vegetation through the plant's root system. The plant concentration due to this process can be calculated as

$$C_v^s = C_s B_{iv} \qquad (6\text{-}11)$$

where C_v^s is the plant concentration at harvest (Ci/kg), C_s is the soil concentration (Ci/kg) and B_{iv} is the element specific soil to plant transfer factor. The total concentration from both processes is

$$C_v = C_v^s + C_v^d \qquad (6\text{-}12)$$

Generally, the contribution of C_v^d to C_v is greater than that of C_v^s for atmospherically dispersed radionuclides.

6.4.3 Concentration in Meat and Milk

For a concentration C_v (Ci/kg) in animal feed, the concentration in meat C_f (Ci/kg) can be calculated as

$$C_f = Q_f F_f C_v \qquad (6\text{-}13)$$

where Q_f is the animal's feed consumption (kg/d) and F_f is the feed to meat transfer factor (d/kg). F_f is element dependent and represents the average mean concentration at slaughter for a unit ingestion rate over the animal's lifetime. Most systematic studies of F_f have been made for cattle or other ruminants, although a few measurements for other species also exist (NCRP84). In practice, even the F_f values for beef are often based on colateral data (Bab84).

Similarly for milk, the concentration C_m (Ci/l) can be calculated as

$$C_m = Q_f F_m C_v \qquad (6\text{-}14)$$

where F_m (d/L) is the equilibrium transfer factor to milk and the other parameters are as for (6-13). Although more statistical data are available for F_m than F_f, the authors of NCRP84 note that the estimation of transfer coefficients to animal products is a subject needing both integration and better documentation.

6.4.4 Summary

Radionuclide intake through the food chain depends upon both the concentration in food and human usage. The concentration in food depends upon the food source; use of foods grown in proximity to the release location, the fraction of an individual's food that is home produced, and other factors can strongly influence the significance of the food pathway. Unfortunately, generally useful validation studies to quantify the substantial uncertainties in the food chain have not been made. References such as NCRP84, Ti83, Mi82, and Li79 cite ranges for some parameters and make limited model uncertainty estimates but do not make quantitative evaluations of the uncertainties for the ingestion pathway taken as a whole.

EPA has chosen a factor of 10 as a reasonable upper bound for the uncertainty in both the deposition rate model and the calculated intake from eating food containing deposited radionuclides. Assuming that the two factors are independent, uncorrelated, and correspond to the 2 sigma values for a log normal distribution, the combined uncertainty for the pathway (deposition and intake of radionuclides from food) is a factor of 26.* EPA has rounded this value to 30 as an estimate of the overall food pathway uncertainty factor.

It is useful to put this uncertainty in context, accepting the premise that the ingestion pathway estimates should be considered reasonable even if their uncertainty does not admit precise quantification. Table 6.4-1 of Volume II of the BID shows that for two elemental phosphorus plants, the portion of the risk due to the ingestion pathway was 0.7 percent for one plant and 0.5 percent for the other. Even a factor of 30 increase in the ingestion pathway risk would not make it a significant fraction of the total risk from all pathways. Fortunately, the food pathway has not proved to be a significant part in assessing the total health risks of radionuclides in air and hence the large uncertainties associated with the food pathway do not limit the overall uncertainty.

6.5 Calculating the Environmental Concentration of Radionuclides: The AIRDOS-EPA Code

6.5.1 Introduction

Environmental concentrations of radionuclides calculated by EPA may be site specific, meaning that available data relevant for the site are incorporated into the assessment. Or an assessment may be generic, that is, an assessment of a hypothetical facility at a location considered an appropriate possibility for such a facility class. Frequently, EPA performs site-specific assessments for existing facilities, e.g., a national laboratory. In addition, EPA often employs generic assessments in evaluating alternative sitings for a proposed facility or assessing a widespread class of facilities, e.g., industrial coal-burning boilers.

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*$\exp[2 \ln^2 (10)]^{1/2} = 26$

In any case, EPA makes both individual and collective (population) assessments. The purpose of the individual assessment is to assess the doses and lifetime risk to individuals living near a facility. EPA's assumption is that these individuals reside a substantial portion of their lives at the same location and that their exposures extend from infancy on through adulthood. The doses and risks calculated are expectation values, i.e., the estimates are intended to be typical for a person living a long period of time under the assessed conditions. EPA's collective (or population) assessments evaluate doses and risks to a population that may be regional (typically up to 80 km distant), long-range (e.g., the conterminous United States), or worldwide as appropriate. The risk is usually expressed as the expected number of premature deaths in the population per year of facility operation.

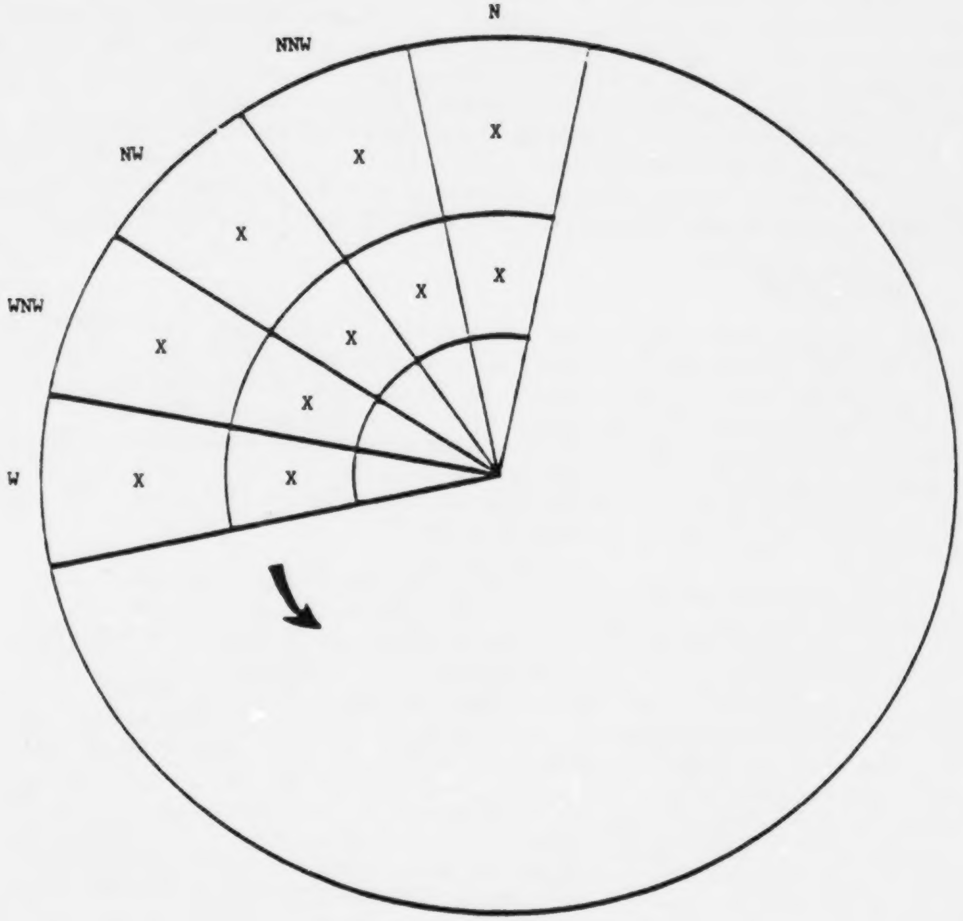
6.5.2 AIRDOS-EPA

EPA has used the AIRDOS-EPA code (Mo79) to calculate environmental concentrations resulting from radionuclide emissions into air. The results of this analysis are estimates of air and ground surface radionuclide concentrations; intake rates via inhalation of air; ingestion of radioactivity via meat, milk, and fresh vegetables. The atmospheric and terrestrial transport models used in the code, their implementation, and the applicability of the code to different types of emissions are described in detail in Mo79.

AIRDOS-EPA calculates atmospheric dispersion for radionuclides released from one to six stacks or area sources. Radionuclide concentrations in meat, milk, and fresh produce are estimated by coupling the deposition rate output of the atmospheric dispersion models with the Regulatory Guide 1.109 (NRC77) terrestrial food chain models. Radionuclide concentrations for specified distances and directions are calculated for the following exposure pathways: (1) immersion in air containing radionuclides, (2) exposure to ground surfaces contaminated by deposited radionuclides, (3) inhalation of radionuclides in air, and (4) ingestion of food in the area. The code may be used to calculate either annual individual exposures or annual population exposures at each grid location. For either option, AIRDOS-EPA output tables summarize air concentrations and surface deposition rates as well as the intakes and exposures for each location. In addition, working level exposures are calculated and tabulated for evaluating the inhalation of short-lived progeny of radon-222.

Assessment Grid

AIRDOS-EPA has provision for either a rectangular or a circular calculational grid. The customarily used circular grid (see Figure 6.5-1) has 16 directions proceeding counterclockwise from north to north-northeast. The user chooses the grid distances. Generally, successive distances are chosen with increasing spacing. It is



X - Assessment grid locations at up to 20 distances (2 shown) and 16 directions (5 shown)

Figure 6.5-1. Circular grid system used by AIRDOS-EPA.

important to realize that the calculational grid distances and the set of distances associated with population and food production data are one and the same. Hence, the concentration calculated for each grid distance must be the appropriate average value for the corresponding range of distances covered by the population and agricultural data. Choosing a suitable set of grid distances may require different compromises of convenience for different assessments and may be different for individual and collective assessments of the same facility.

Environmental Accumulation Time

An AIRDOS-EPA assessment is based on what can be viewed as a snapshot of environmental concentrations after the assessed facility has been operating for some period of time. The choice of an environmental accumulation time affects only those pathways dependent on terrestrial concentrations, i.e., ground surface exposure and food intakes. Usually, the accumulation time for an individual assessment is chosen to be consistent with the expected life of the facility (or 100 years when a similar facility might be expected to replace the present one at the end of its useful life). For collective assessments, 100 years is customarily used.

Source Considerations

Point sources are characterized by their physical height and, when desired, the parameters to calculate buoyant or momentum plume rise using Brigg's (Br69) or Rupp's (Ru48) formulations respectively. Alternatively, a fixed plume rise may be specified for each Pasquill-Gifford atmospheric stability class A through G.

The area source model is similar to that of Culkowski and Patterson (Cu76) and transforms the original source into an annular segment with the same area. At large distances, the transformed source approaches a point source at the origin, while at distances close to the origin it approaches a circle with the receptor at its center.

Building wake effects and downwash are not included in the AIRDOS-EPA models. The same type of rise calculation (buoyant, momentum, or fixed) is used for all sources. As many as six sources may be assessed, but for calculational purposes they are all considered to be co-located at the origin of the assessment grid.

Radionuclide Releases

Releases for up to 36 radionuclides may be specified for AIRDOS-EPA. Each release is characterized by the radionuclide name, effective decay constant during dispersion, precipitation scavenging coefficient, deposition velocity, and settling velocity as well as the annual activity release for each source. Decay products that are significant for the assessment of a radionuclide must be included in the list of releases. There is no explicit method for calculating radionuclide ingrowth during atmospheric dispersion in AIRDOS-EPA.

Parameters such as particle size, respiratory clearance class, and gastrointestinal absorption factor (f_1) are passed on for use in the DARTAB (Be81) dose and risk assessments as described in the Appendices to Chapters 7 and 8.

The approach ORP has used for calculating a precipitation scavenging coefficient is based on Slinn's (Sl77) equation (32):

$$\lambda_{sc} = \frac{c\ J_o\ E(a,R_m)}{R_m} \qquad (6\text{-}7)$$

where λ_{sc} is the scavenging coefficient, c is a constant (Slinn uses 0.5), J_o is the rainfall rate, and E is the collection efficiency for a particle of radius a by drops of characteristic radius R_m. Slinn (Sl77, p. 23) considers the effects of dry deposition and interprets Dana and Wolf's (Daa68, Wo69, Dab70) data as supporting a value for E of 0.2, essentially independent of particle size. Adopting Slinn's typical value of R_m for a frontal rain (0.3 mm) and selecting a long-term average value of 1000 mm/yr ($3.16\text{x}10^{-5}$ mm/s) for J_o, we obtain:

$$\lambda_{sc} = \frac{0.5\ 3.16\text{x}10^{-5}\ 0.2}{0.3} \qquad (6\text{-}8)$$

$$= 1.05\text{x}10^{-5}\ s^{-1}$$

This value has been rounded to $10^{-5}\ s^{-1}$ as a working value for the precipitation scavenging coefficient and then scaled according to the annual precipitation at the assessment location for use in AIRDOS-EPA. There is substantial uncertainty in interpreting environmental scavenging data; this estimate is clearly an order of magnitude one. The EPA scaling procedure reflects the premise that the variation of rainfall from one location to another is more one of rain frequency than of intensity during rainfall episodes.

Dispersion

Wind and stability class frequencies for each direction are the primary data for calculating atmospheric dispersion. The required data for AIRDOS-EPA are calculated from a joint frequency distribution of wind speed and atmospheric stability class for each direction.

Inasmuch as the assessments require long-term average dispersion values, the sector-averaged Gaussian plume option is used. The vertical dispersion parameter (σ_z) is calculated using Brigg's formulas (Gi76). Vertical dispersion is limited to the region between the ground and a mixing depth lid. The harmonic mean of Holzworth's (Hoa72) morning and afternoon mixing depths is customarily employed for this value, that is,

$$h_\ell = 2 \frac{\ell_a \ell_p}{\ell_a + \ell_p} \quad , \qquad (6\text{-}9)$$

where ℓ_a and ℓ_p are respectively the morning and afternoon mixing depths and h_ℓ is their harmonic mean. At large distances, the concentration is uniform between the ground and the lid.

Deposition Rate

AIRDOS-EPA models both dry and wet deposition processes. Resuspension, the reintroduction of deposited material into the atmosphere, is not modeled in AIRDOS-EPA. The dry deposition rate is the product of the deposition velocity and the near ground level air concentration while the wet deposition rate is the product of the precipitation scavenging coefficient and the vertically integrated air concentration. Wet deposition decreases monotonically with distance and is independent of the effective release height of the source, while the effect of source height can be significant for dry deposition. For locations close to an elevated source, wet deposition can provide the principal source of radionuclide exposure. Concentrations are adjusted for depletion due to deposition at each downwind distance.

Ground Surface Concentration

AIRDOS-EPA calculates the ground surface concentration from the total (dry plus wet) deposition rate. The soil concentration is calculated by dividing this value by the effective agricultural soil surface density (kg/m^2). Both concentrations are calculated for the end of the environmental accumulation time t_b and can include the ingrowth from deposited parent radionuclides as well as removal due to radiological decay and environmental processes such as leaching.

Ingrowth from a parent radionuclide is calculated using a decay product ingrowth factor. The ingrowth factor is the equivalent deposition rate for a unit deposition rate of the parent radionuclide. For example, the ingrowth factor for lead-210 as a parent of polonium-210 would be calculated by determining the concentration of polonium-210 at time t_b due to a unit deposition rate of lead-210 and dividing it by the corresponding concentration for a unit deposition rate of polonium-210. These ingrowth factors must be calculated in advance of running AIRDOS-EPA and are dependent on both the accumulation time t_b and the soil removal constants for the nuclides in the radionuclide chain (lead-210, bismuth-210, and polonium-210 in this case).

Concentrations in Food

Radionuclide concentrations in food are calculated using essentially the same model as in NRC Regulatory Guide 1.109 (NRC77). Changes from that model include consideration of environmental removal from the root zone, and separate values for food and pasture crops of the interception fraction, areal yield, and soil-to-plant transfer values. Concentration calculations for meat and milk use the same models as the Regulatory Guide model.

There are numerous parameters in the terrestrial pathways model. Appendix A of Volume II of the BID contains tables of values used in these assessments.

Population and Agricultural Data

For a collective (population) assessment, population and agricultural data for each grid location must be provided. EPA uses the 1970 census enumeration district data to calculate population distributions. AIRDOS-EPA calculates the collective assessment for agricultural products based on consumption by the assessment area population. The assessment can be based on agricultural production by choosing utilization factors large enough to ensure that all items produced are consumed.

Food Utilization Factors

In addition to the consumption rate for different food categories (leafy vegetables, other produce, meat, and milk), the user may specify the fraction of vegetables, meat, and milk that are (1) home grown, (2) produced in the assessment area, or (3) imported from outside the assessment area. Those in the third category are considered to contain no radionuclides. Those from the second category have the average concentration for that category produced within the assessment area, while concentrations for the first category are those that would occur at each grid location. Appendix A of Volume II of the BID provides some typical food source fractions for urban and rural assessment areas. Note that if the assessment considers food to be only home grown or imported from outside the assessment area, then the actual quantity of food produced at each location is not relevant to the assessment. Experience has shown that the ingestion doses and risks for the nearby individual are usually dominated by the radionuclide intake from home grown food and hence there is generally no significant difference between assuming that food that is not home grown is obtained from the assessment area or is imported from outside the assessment area.

Special Radionuclides

Special consideration is given to the radionuclides hydrogen-3 (tritium), carbon-14, and radon-222. The specific activity of tritium in air (pCi/g of H_2O) is calculated for an absolute humidity of 8 mg/m^3 (NRC77). Etnier (Et80) has calculated average absolute humidities for over 200 U.S. locations. The 8 mg/m^3 value would be within a factor of 2 for most of them. The specific activity of atmospheric carbon-14 (pCi/g of C) is calculated for a CO_2 concentration of 330 ppm by volume (Ki78). Concentrations of these nuclides in vegetation are calculated on the assumption that the water and carbon content in vegetation are from the atmosphere and have the same specific activity as in the atmosphere. The radon-222 concentration in air is replaced by its short-lived decay product concentration in working level units using a fixed equilibrium fraction (typically 0.7 for calculating population health risks).

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Chapter 7: RADIATION DOSIMETRY

7.1 Introduction

Radionuclides transported through the environment may eventually reach people. This contact occurs through either external exposure to radioactive air, water, and ground surfaces or internal exposure from inhaling or ingesting radioactive air, water, or food. Individuals in the population may absorb energy emitted by the decaying radionuclides. The quantification of this absorbed energy is dosimetry. This chapter describes the dosimetric models for internal and external exposures, the EPA procedure for implementing the dosimetric equations associated with the models, and the uncertainties in dosimetric calculations.

Mathematical models are used to calculate doses to specific human body organs. The models account for the amount of radionuclides entering the body, the movement of radionuclides through the body, and the energy deposited in organs or tissues resulting from irradiation by the radionuclides that reach the tissue. These models provide the basis for the computer codes, RADRISK and DARTAB, which EPA uses to calculate doses and dose rates. (See Addendum A.)

Uncertainties in dosimetric calculations arise from assumptions of uniform distribution of activity in external sources and source organs and assumptions concerning the movement of the radionuclides in the body. The uncertainties associated with dosimetric calculations are difficult to quantify because the data available for determining distribution for the parameters used in the models are usually insufficient. The major source of uncertainty in dosimetry is the real variation in parameter values among individuals in the general population while doses and dose rates are calculated for a "typical" member of the general population. The three sources of dosimetric uncertainty assessed by EPA are: individual variation, age, and measurement errors. The effects of uncertainty analysis on the dose estimates for the general population are discussed in greater detail in Section 7.6.

7.2 Definitions

7.2.1 Activity

Radioactive decay is a process whereby the nucleus of an atom emits excess energy. The emission of this energy is referred to as radioactivity. The "activity" of a radioactive material is characterized by the number of atoms that emit energy, or disintegrate, in a given period of time. The unit of activity used in this report is the picocurie (pCi), which equals 2.22 disintegrations per minute. The excess energy is normally emitted as charged particles moving at high velocities and photons. Although there are many types of emitted radiations, or particles, only three are commonly encountered in radioactive material found in the general environment: alpha radiation (nuclei of helium atoms), beta radiation (electrons), and gamma radiation (photons).

The primary mechanism for radiation damage is the transfer of kinetic energy from the moving alpha and beta particles and photons to living tissue. This transfer leads to the rupture of cellular constituents resulting in electrically charged fragments (ionization). Although the amount of energy transferred is small in absolute terms, it is enough to disrupt the molecular structure of living tissue, and, depending on the amount and location of the energy release, lead to the risk of radiation damage.

7.2.2 Exposure and Dose

The term "exposure" denotes physical contact with the radioactive material. The term "dose" refers to the amount of energy absorbed per gram of absorbing tissue as a result of the exposure. An exposure, for example, may be acute, i.e., occur over a short period of time, while the dose, for some internally deposited materials, may extend over a long period of time.

The dose is a measure of the amount of energy deposited by the alpha and beta particles or photons and their secondary radiations in the organ. The only units of dose used in this chapter are the rad--defined as 100 erg (energy units) per gram (mass unit)--and the millirad (mrad), which is one one-thousandth of a rad. The rad represents the amount, on average, of potentially disruptive energy transferred by ionizing radiation to each gram of tissue. Because it is necessary to know the yearly variation in dose for the calculations described in this report, the quantity used will be the average annual dose (or dose rate) in rad or millirad (per year).

7.2.3 External and Internal Exposures

Radiation doses may be caused by either external or internal exposures. External exposures are those caused by radioactive materials located outside the body, such as irradiation of the body by radioactive material lying on the ground or suspended in the air. Internal exposures are caused by radioactive material that has entered the body through the inhalation or consumption of radioactive material. Having once entered the body, the contaminant may be transmitted to other internal organs and tissues.

The external exposures considered in this report are those resulting from irradiation of the body by gamma rays only. Gamma rays (high energy photons) are the most penetrating of those radiations considered and external gammas may normally contribute to the radiation dose affecting all organs in the body. Beta particles (electrons), which are far less penetrating, normally deliver their dose to, or slightly below, the unshielded surface of the skin and are not considered because their impact is small, particularly on clothed individuals. Alpha particles (helium nuclei), which are of major importance internally, will not penetrate unbroken skin and so are also excluded from the external dose calculations. The internal exposures considered in this report originate from all three types of radiation.

7.2.4 Dose Equivalent

Different types of charged particles differ in the rate at which their energy is transferred per unit of length traveled in tissue, a parameter called the linear energy transfer (LET) of the particle. Beta particles generally have a much lower LET than alpha particles. Alpha particles are more damaging biologically, per rad, than gamma rays and beta particles. In radiation protection, this difference is accounted for by multiplying the absorbed dose by a modifying factor, Q, the quality factor, to obtain a dose equivalent. The quality factor is intended to correct for the difference in LET of the various particles. At present, the International Commission on Radiological Protection (ICRP77) recommends the values $Q=1$ for gamma rays and beta particles and $Q=20$ for alpha particles. The units for the dose equivalent, corresponding to the rad and millirad, are rem and millirem. Thus, dose equivalents for gamma rays and beta particles are numerically equal to the dose since the dose equivalent (mrem) = $(Q=1)$ x dose (mrad) while alpha dose equivalents are twenty times as large, dose equivalent (mrem) = $(Q=20)$ x dose (mrad).

7.3 Dosimetric Models

The radiation dose has been defined, in 7.2.2, as the amount of energy absorbed per unit mass of tissue. Calculation of the dose requires the use of mathematical models such as that shown later in equation 7-2. In this equation, the amount of activity ingested, I, is multiplied by the fraction, f_1, going to the blood, and the fraction, f_2', going to a specific tissue. E is the amount of energy absorbed by the tissue for each unit of activity so that the product of all these factors divided by the mass of the tissue is, by definition, the radiation dose. The remaining term, $[1-e^{-\lambda t}]/\lambda$, indicates how the activity deposited in the tissue changes with time. All these factors together yield the dose rate. A more comprehensive description of the equations used is given in Addendum A.

7.3.1 Internal Doses

Any effort at calculating dose and risk must, of necessity, involve the use of models. In its simplest form, a model is a mathematical representation of a physical or biological system. If, for example, the amount of radioactive material in an organ is measured at several times, a graph of the activity in the organ, such as that in Figure 7.3-1, is

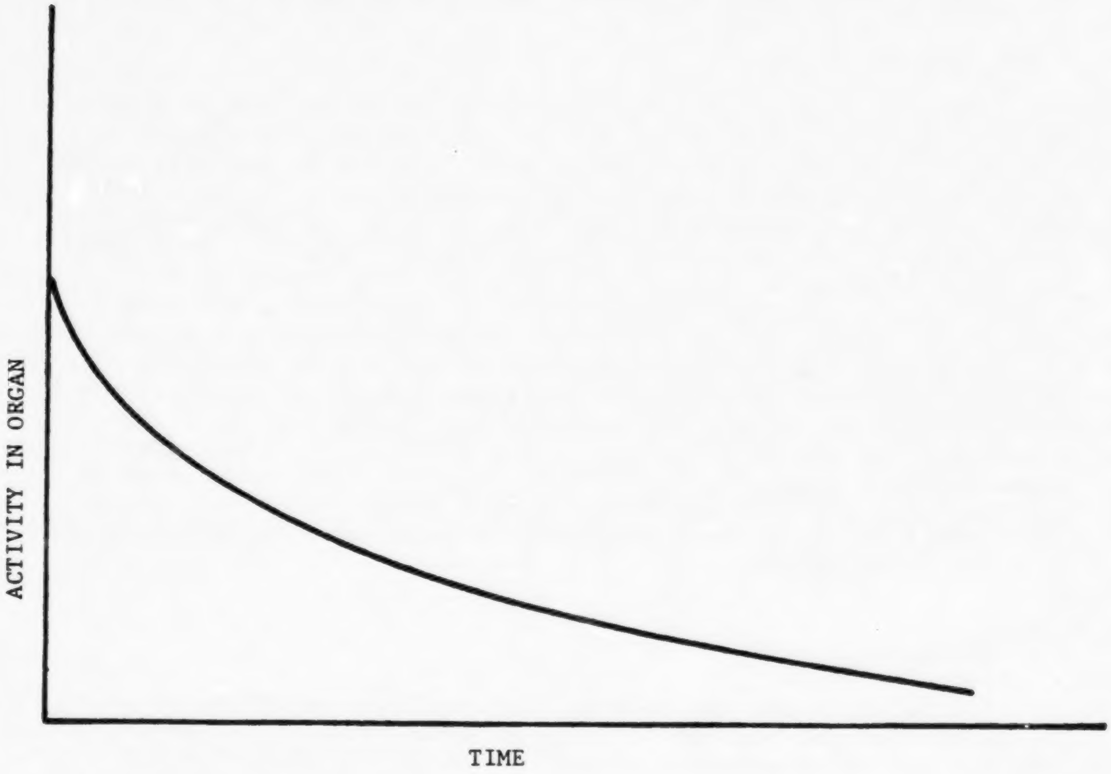

Figure 7.3-1. Typical pattern of decline of activity of a radionuclide in an organ, assuming an initial activity in the organ and no additional uptake of radionuclide by the organ (ORNL81).

obtained. In the simplest case, analysis of these data may indicate that the fraction of the initial activity, R, retained in the organ at any time, t, is given by an equation of the form

$$R = e^{-\lambda t} \qquad (7\text{-}1)$$

where λ is the elimination rate constant. (More generally, it may require the sum of two or more exponential functions to properly approximate the decrease of radioactivity in the organ. This may be interpreted physically as indicating the existence of two or more "compartments" in the organ from which the nuclide leaves at different rates.)

The elimination rate constant, λ, is the sum of two terms, which may be measured experimentally, one inversely proportional to the biological clearance half-life and the other inversely proportional to the radioactive half-life. The effective half-life, $t_{1/2}$, for these processes is the time required for one-half of the material originally present to be removed.

If radionuclides are generally found to follow this behavior, then this equation may be used as a general model for the activity in an organ following deposition of any initial activity. In general, the models used by EPA are those recommended by the International Commission on Radiological Protection (ICRP79) and are documented in detail in the cited reference. A brief description of each model is given below as an aid to understanding the material presented in the balance of this chapter.

As mentioned earlier, all radiations--gamma, beta, and alpha--are considered in assessing the doses resulting from internal exposure, that is, exposure resulting from the inhalation or ingestion of contaminated material. Portions of the material inhaled or ingested may not leave the body for a considerable period of time (up to decades); therefore, dose rates are calculated over a corresponding time interval.

The calculation of internal doses requires the use of several models. The most important are the ICRP lung model, depicted in Figure 7.3-2, and the gastrointestinal (GI) tract model shown in Figure 7.3-3. The lung model is comprised of three regions, the nasopharyngial (N-P), the tracheobronchial (T-B), and the pulmonary (P) regions. A certain portion of the radioactive material inhaled is deposited in each of the three lung regions (N-P, T-B, and P) indicated in Figure 7.3-2. The material is then cleared (removed) from the lung to the blood and gastrointestinal tract, as indicated by the arrows, according to the specified clearance parameters for the clearance class of the inhaled material.

Deposition and clearance of inhaled materials in the lung are controlled by the particle size and clearance class of the material. The particle size distribution of the airborne material is specified by

COMPARTMENT		CLASS					
		D		W		Y	
		T	F	T	F	T	F
N-P	a	0.01	0.5	0.01	0.1	0.01	0.01
$(D_3 = 0.30)$	b	0.01	0.5	0.4	0.9	0.4	0.99
T-B	c	0.01	0.95	0.01	0.5	0.01	0.01
$(D_4 = 0.08)$	d	0.2	0.05	0.2	0.5	0.2	0.99
	e	0.5	0.8	50	0.15	500	0.05
P	f	n.a.	n.a.	1.0	0.4	1.0	0.4
$(D_5 = 0.25)$	g	n.a.	n.a.	50	0.4	500	0.4
	h	0.5	0.2	50	0.05	500	0.15
L	i	0.5	1.0	50	1.0	1000	0.9

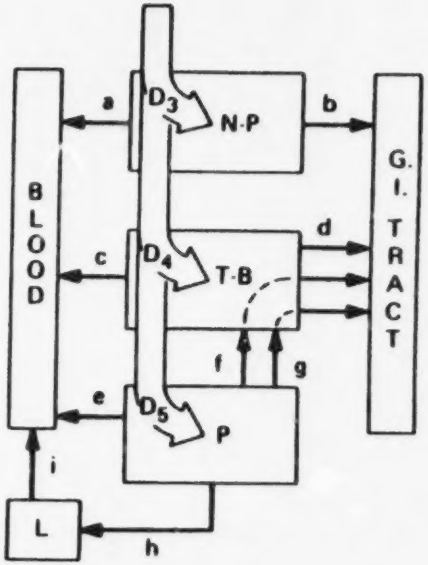

Figure 7.3-2. The ICRP Task Group lung model for particulates.

The columns labeled D, W, and Y correspond, respectively, to rapid, intermediate, and slow clearance of the inspired material (in days, weeks, or years). The symbols T and F denote the biological half-time (days) and coefficient, respectively, of a term in the appropriate retention function. The values shown for D_3, D_4, and D_5 correspond to activity median aerodynamic diameter AMAD = 1 μm and represent the fraction of the inspired material depositing in the lung regions.

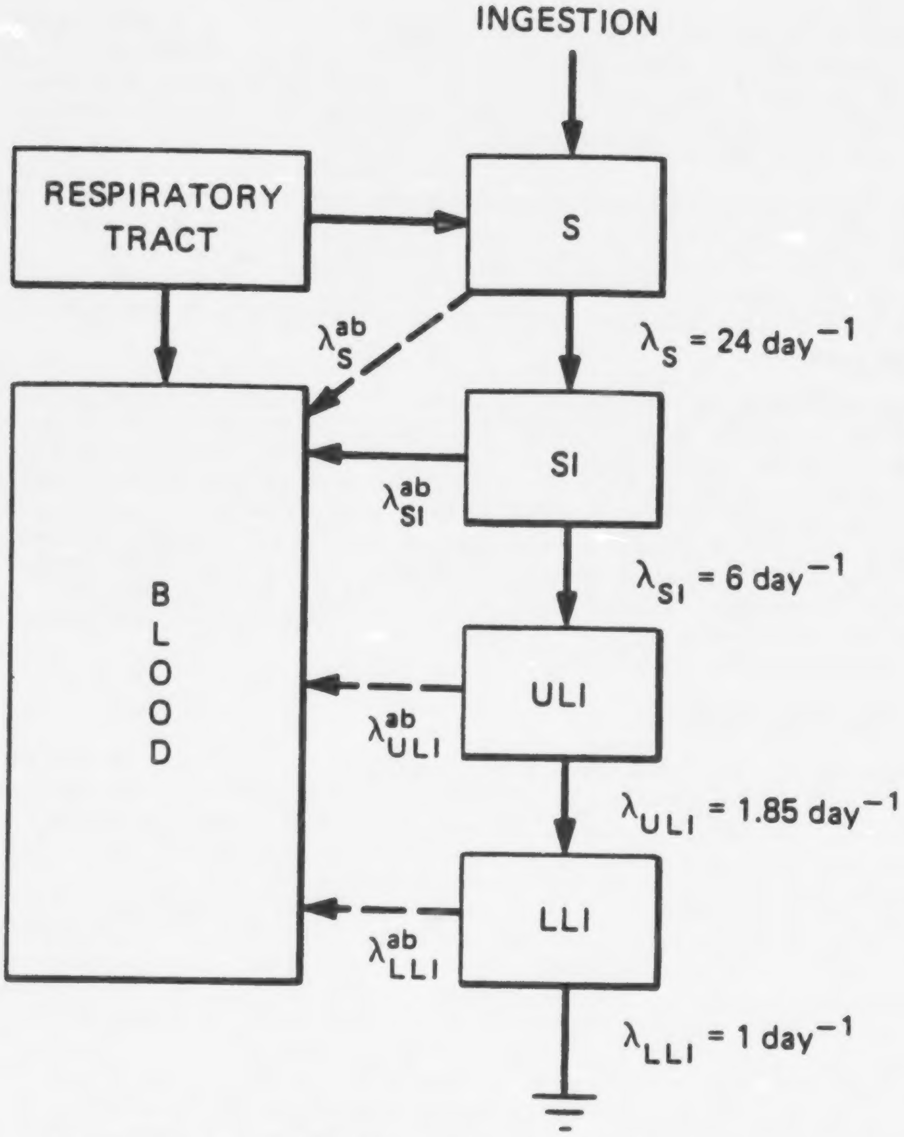
INGESTION
RESPIRATORY TRACT
S
λ_S^{ab}
$\lambda_S = 24\ \text{day}^{-1}$
SI
λ_{SI}^{ab}
$\lambda_{SI} = 6\ \text{day}^{-1}$
BLOOD
ULI
λ_{ULI}^{ab}
$\lambda_{ULI} = 1.85\ \text{day}^{-1}$
LLI
λ_{LLI}^{ab}
$\lambda_{LLI} = 1\ \text{day}^{-1}$

Figure 7.3-3. Schematic representation of radioactivity movement among respiratory tract, gastrointestinal tract, and blood.

S = stomach
SI = small intestine
ULI = upper large intestine
LLI = lower large intestine
λ = elimination rate constant

giving its Activity Median Aerodynamic Diameter (AMAD) in microns (one micron equals 10^{-6} meters). Where no AMAD is known, a value of 1.0 micron is assumed. Clearance classes are stated in terms of the time required for the material to leave the lung, that is, Class D (days), Class W (weeks), and Class Y (years).

The gastrointestinal tract model consists of four compartments, the stomach (S), small intestine (SI), upper large intestine (ULI), and lower large intestine (LLI). However, it is only from the small intestine (SI) that absorption into the blood is considered to occur. The fraction of material that is transferred into blood is denoted by the symbol f_1.

Radionuclides may be absorbed by the blood from either the lungs or the GI tract. After absorption by the blood, the radionuclide is distributed among body organs according to fractional uptake coefficients, denoted by the symbol f_2'. Since the radioactive material may be transported through the body, dose rates are calculated for each organ or tissue affected by using a model of the organ that mathematically simulates the biological processes involved. The general form of the model for each organ is relatively simple. It postulates that the radioactive material which enters the organ is removed by both radioactive decay and biological removal processes.

7.3.2 External Doses

The example just described for modeling the activity of a radionuclide in an organ pertains to estimating doses from internal exposure. In contrast, the external immersion and surface doses are calculated as follows. First, the number of photons reaching the body is determined. The model used here is a set of equations governing the travel of photons (gamma radiation) in air. The simplifying assumptions used in these calculations are that the medium (air) is an infinite half-space and is the only material present. This makes the calculation relatively straightforward. In the second portion of the calculation, the photons reaching the body are followed through the body using a "Monte Carlo" method. The "phantoms", i.e., the models of the body, are those used by the Medical Internal Radiation Dose Committee (MIRD69). The Monte Carlo method is a procedure in which the known properties of the radiation and tissues are employed to trace (simulate) the paths of a large number of photons in the body. The amount of energy released at each interaction of the radiation with body tissues is recorded and, thus, the dose to each organ or tissue is estimated by evaluating a large number of photon paths.

7.3.3 Effects of Decay Products

In calculating doses from internal and external exposures, the occurrence of radioactive decay products (or daughters) must be considered for some radionuclides. When an atom undergoes radioactive decay, the new atom created in the process may also be radioactive and may contribute to the radiation dose. Although these decay products may be treated as independent radionuclides in external exposures, the decay products of each parent must be followed through the body in internal exposures. The decay product contributions to the dose rate are included in the dose calculations, based on the metabolic properties of the element and the organ in which they occur.

7.3.4 Dose Rate Estimates

For each external and internal exposure, dose rates to each of the organs listed in Table 7.3-1 are calculated for each radioisotope. These organ dose rates serve as input to the life table calculations described in Chapter 8.

Table 7.3-1. Organs for which dose rates are calculated

Red bone marrow	Intestine
Bone	Thyroid
Lung	Liver
Breast	Urinary tract
Stomach	Other(a)
Pancreas	

(a)Esophagus, lymphatic system, pharynx, larynx, salivary gland, brain.

7.4 EPA Dose Calculation

7.4.1 Dose Rates

The models described in Section 7.2 are used by EPA to calculate radiation dose rates resulting from internal and external exposures to radioactive materials. A more complete description of the methodology, equations, and parameters used is given in Du84, ORNL80, and ORNL81. EPA has adopted two refinements to the ICRP-recommended protocol for these calculations. The first is to track the movement of internally produced radioactive daughters by assuming that their movement is governed by their own metabolic properties rather than those of the parent. Although not enough information is available to allow a rigorously defensible choice, this appears to be more accurate for most organs and nuclides than the ICRP assumption that daughters behave exactly as the parent. In the second departure from ICRP recommendations, age-dependent values of the parameters governing the uptake of transuranic nuclides have been taken from two sources, deemed appropriate to the general population, the National Radiological Protection Board (NRPB82) and the EPA transuranic guidance document (EPA77).

The internal dose equations given by ICRP may be used to calculate either radiation doses (rad), i.e., the total dose over a given time period, or radiation dose rates (rad/yr), i.e., the way in which the dose changes with time after intake. The summation of the dose rates is, of course, the total dose. EPA calculates dose rates rather than doses, because EPA considers age when assessing the effects of radiation on the population.

External irradiation does not result in any residual internal material. Therefore, external dose rates to a given organ are constant. That is, the dose rate caused by a given amount of radionuclide present in air or on a ground surface becomes zero when the radionuclide is removed.

The calculation of dose rates, rather than integrated doses, allows the use of age-dependent metabolic parameters more appropriate to the general population to be taken into account. In the vast majority of cases, however, there is not now sufficient information available to make such calculations. The major exception to this is exposure to radon, in which EPA uses age-dependent exposure parameters. Because most of the data available for radon are in terms of exposure, no doses are calculated for this gaseous element. Radon assessments are discussed in detail in Chapter 8. The effect of using age-dependent metabolic parameters is discussed in Section 7.5.2 for some radionuclides for which sufficient information is available.

7.4.2 Exposure and Usage

The ICRP dosimetric equations used by EPA are linear, i.e., an intake of 10 picoCuries will result in dose rates ten times as large as those from an intake of 1 picoCurie. In similar fashion, exposure to 10 times as large an air or ground surface concentration will increase the external doses by a factor of ten. EPA uses this linearity to avoid having to calculate radiation dose rates for a range of concentrations. The standard EPA procedure is to use unit intakes of 1 pCi/yr and air and ground surface concentrations of 1 pCi/cm^3 and 1 pCi/cm^2, respectively. The doses for other intakes and concentrations may then be scaled up or down as required.

In most cases, it is necessary to make certain assumptions regarding the exposure conditions in order to perform an assessment. EPA calculates dose rates for lifetime exposure to the unit intakes and concentrations. Chapter 8 describes the different ways in which these rates can be applied. In addition, the exposure assessment will usually depend on other usage conditions assumed for the exposures.

Thus, for the general population, EPA assumes a breathing rate, using ICRP-recommended values (ICRP75), based on 8 hours of heavy activity, 8 hours of light activity, and 8 hours of rest per day. When required, EPA uses a drinking water intake of 2 liters per day. The quantities of food ingested are compiled from a variety of sources. Because there may be insufficient data for some types of food, it may be necessary to combine or substitute types in some instances. More complete details on the values used for the ingestion of foodstuff types are given in Appendix A of Volume 2.

7.5 Uncertainty Analysis

Uncertainty, in the dose, refers to the manner in which the calculated dose changes when the parameters used in the calculation (intakes, metabolic factors, organ sizes, etc.) are changed. The uncertainty associated with the dosimetric calculations is extremely difficult to quantify because the term "uncertainty analysis" implies a knowledge of parameter distributions that is usually lacking. Internal doses, for example, depend on the parameters used to characterize the physiological and metabolic properties of an individual, while external doses must consider parameters such as organ size and geometry for a particular individual. The data available for most of these parameters is not sufficient to define the form of the parameter distribution. The major source of uncertainty in calculating the dose to a distinct individual, however, in most instances, does not result from errors in measuring the parameters but from the real variation in parameter values among individuals in the general population. Thus, a calculated dose is thought to be representative of a "typical" member of the general population and is probably reasonably precise for some large segment of that population.

The basic physiological and metabolic data used by EPA in calculating radiation doses are taken from the ICRP Report of the Task Group on Reference Man (ICRP75) and from the ICRP Limits for Intakes of Radionuclides by Workers (ICRP79). The "Reference Man" report is the most comprehensive compilation of data available on the intake, metabolism, internal distribution, and retention of radioisotopes in the human body. Its major purpose, however, is to "define Reference Man, in the first instance, as a typical occupational individual", although differences with respect to age and sex are indicated in some instances.

The limitations inherent in defining Reference Man, and in estimating uncertainties due to variations in individuals in the general population, are recognized by the Task Group (ICRP75):

> "The Task Group agreed that it was not feasible to define Reference Man as an 'average' or a 'median' individual of a specified population group and that it was not necessary that he be defined in any such precise statistical sense. The available data certainly do not represent a random sample of any specified population. Whether the sample is truly representative of a particular population group remains largely a matter of judgement which cannot be supported on the basis of statistical tests of the data since the sampling procedure is suspect. Thus the Task Group has not always selected the 'average', or the 'median', of the available measurements in making its selection, nor has it attempted to limit the sample to

> some national or regional group and then seek an average or median value. However, the fact that Reference Man is not closely related to an existing population is not believed to be of any great importance. If one did have Reference Man defined precisely as having for each attribute the median value of a precisely defined age group in precisely limited locality (e.g., males 18-20 years of age in Paris, France, on June 1, 1964), these median values may be expected to change somewhat with time, and in a few years may no longer be the median values for the specified population. Moreover, the Reference Man so defined would not have this relation to any other population group unless by coincidence. To meet the needs for which Reference Man is defined, this precise statistical relationship to a particular population is not necessary. Only a very few individuals of any population will have characteristics which approximate closely those of Reference Man, however he is defined. The importance of the Reference Man concept is that his characteristics are defined rather precisely, and thus if adjustments for individual differences are to be made, there is a known basis for the dose estimation procedure and for the estimation of the adjustment factor needed for a specified type of individual."

With respect to the dosimetric calculations performed by EPA to assess the impact of radioactive pollutants on a general population, three sources of uncertainty should be considered:

(1) that due to the variation in individual parameters among adults in the general population

(2) that due to the variation in individual parameters with age

(3) that due to experimental error in the determination of specific parameters

Each of these sources of uncertainty is discussed in this section. As noted above, the data required to perform a rigorous sensitivity analysis are lacking, and a form of uncertainty analysis called sensitivity analysis is employed. The sensitivity analysis consists of substituting known ranges in the parameters for the recommended value and observing the resulting change in the calculated dose.

7.5.1 Dose Uncertainty Resulting from Individual Variation

This section discusses the uncertainty in calculated radiation doses occasioned by differences in physical size and metabolism among individuals in the general population. In order to investigate the effects of individual differences in intake, size, and metabolism, it is necessary to consider the form of the equation used to calculate radiation dose rates. Equation 7-2 is a simplified form of the one used by EPA to represent the ingestion of radioactive materials.

$$\dot{D}(t) = c\ I\ f_1\ f_2' \frac{E}{m} \frac{1}{\lambda} [1-e^{-\lambda t}] \qquad (7\text{-}2)$$

where $\dot{D}$ is the dose rate
I is the intake of radioactivity
f_1 is the fraction of I transferred to blood after ingestion
f_2' is the fraction transferred to an organ from the blood
m is the mass of the organ
λ is the elimination constant, which denotes how rapidly the activity is removed from the organ
E is the energy absorbed by the organ for each radioactive disintegration
c is a proportionality constant.

For simplicity, we will assume that dose rates at large times, t, are to be studied so that the term in the bracket is approximately unity.

Although the actual equations used are considerably more complicated because they must describe the lung model and the GI tract, and also treat all radioactive progeny, the essential features of the uncertainty in dose calculation are reflected in the terms of Equation (7.2). The sensitivity of the dose to each of the terms in the equation may be studied by substituting observed ranges of the quantities for the single value recommended by Reference Man. For some of these quantities, as noted below, no range is cited because of insufficient data.

Daily Intake, I

As an example, postulate that the ingestion mode to be calculated is for fluid intakes. The average daily fluid intake is about 1900 ml, with an adult range of 1000 to 2400 for "normal" conditions. Under higher environmental temperatures, this range may be increased to 2840 to 3410 ml. Thus, a dose calculated as 1.9, for example, could range from 1.0 to 2.4.

Transfer Fraction, f_1

The value of the transfer fraction to blood depends on the chemical form of the element under study. One of the most common naturally occurring radionuclides is uranium, which is used here as an example. ICRP79 cites values of f_1 ranging from 0.005 to 0.05 for industrial workers, but notes that a higher value of 0.2 is indicated by dietary data from persons not occupationally exposed. EPA has used the 0.2 value for the general population but, based on the ICRP range above, a calculated dose determination could vary by a factor of 10.0.

Organ Mass, m

The range of organ masses depends primarily on the organ under investigation. For example, reported values for the bloodless lungs range from 461 to 676 grams. Liver weights ranged from 1400 to 2300 grams for adult males and 1200 to 1820 grams for females. Thus, because the organ mass appears in the denominator, calculated lung doses might be expected to vary by a factor of 1.5 and liver doses by a factor of about 2.0. For the radionuclide emissions rulemaking, the uncertainty in the lung mass is the most important consideration, because most of the dose results from inhalation and the lung is usually the principal organ at risk.

Remaining Terms, f_2', λ, E

There are few reported data on the ranges in values to be expected for the remaining variables. They are all quantities which are less directly observable than I, f_1, and m and their influence on the dose calculation can only be estimated. The discussion in Section 7.6 is intended to augment the uncertainty analysis by introducing the results of some direct observations on segments of the general population.

7.5.2 Dose Uncertainty Resulting from Age

The dose rates calculated by EPA are normally based on the metabolism and physical characteristics of Reference Man (ICRP75). These properties may obviously be expected to depend on the age of an individual. Most particularly, for infants and children such factors as breathing rates, liquid and solid intakes, organ size and growth rates, and body geometry are known to vary considerably from adult values. The effect of such changes on the radiation dose also depends on the chemistry of the radioactive element under study. For example, rapid bone growth in children is of more importance when a "bone seeker" such as strontium is considered. Although the data available for most age and chemical element combinations are insufficient to allow estimation of the uncertainty in dose, some organ/element combinations, for which more information is available, are discussed below.

Iodine and the Thyroid

Iodine is rapidly and virtually completely absorbed into the bloodstream following inhalation or ingestion. From the blood, iodine enters the extracellular fluid and quickly becomes concentrated in the salivary, gastric, and thyroid glands. It is rapidly secreted from the salivary and gastric glands, but it is retained in the thyroid for relatively long periods.

The intake and metabolism of iodine have been reviewed extensively (ORNL84a) to develop an age-dependent model for iodine. In the model used here, ingested iodine is assumed to be almost completely absorbed by the blood. The remaining parameters are age dependent and are shown in Table 7.5-1. The fluid intake varies from 0.72 liters per day for a newborn to about 2.0 liters per day for an adult.

These age-dependent parameters may then be used in Equation (7-2) to calculate the dose rate resulting from a constant concentration of iodine in water and air. The resulting curves for the dose rate as a function of age are shown in Figures 7.5-1 and 7.5-2 (note: 1 μCi = 10^6 pCi). These may be compared to the dose rates obtained using Reference Man parameters at all ages, indicated by the dotted lines in the same figures. Thus, for this particular combination of organ and isotope, the total (70 year) dose is seen to increase by about 30 percent for ingestion and 35 percent for inhalation when dependence on age is considered.

Strontium and Bone

Because of the chemical similarities of strontium and calcium, strontium tends to follow the calcium pathways in the body and deposits to a large extent in the skeleton. In fact, the fraction of ingested strontium eventually reaching the skeleton at a given age depends largely on the skeletal needs for calcium at that age, although the body is able to discriminate somewhat against strontium in favor of calcium after the first few weeks of life.

The ICRP model for bone is more complicated than that for the thyroid because it consists of more than one compartment. For purposes of modeling the transport of strontium by the skeleton, it suffices to view the mineralized skeleton as consisting of two main compartments: trabecular (cancellous, porous, spongy) and cortical (compact) bone.

Table 7.5-1. Age-dependent parameters for iodine metabolism in the thyroid

Age (days)	Fractional uptake to thyroid, f_2'	Thyroid mass (g)	Biological half-time in the thyroid (days)
Newborn	0.5	-	15
100	0.40	-	20
365	0.3	1.78	30
1825	0.3	3.45	40
3650	0.3	7.93	50
5475	0.3	12.40	65
7300	0.3	20.00	80

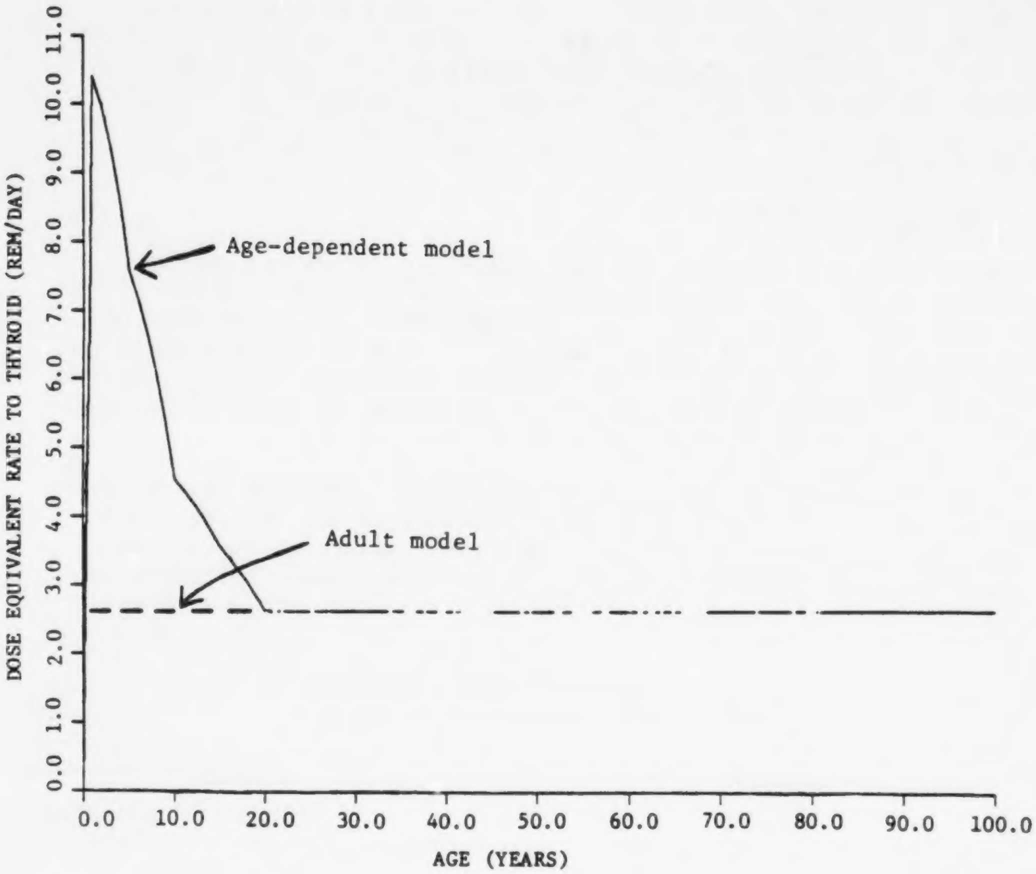

Figure 7.5-1. Dose from chronic ingestion of iodine-131 in water at a concentration of 1 μCi/l.

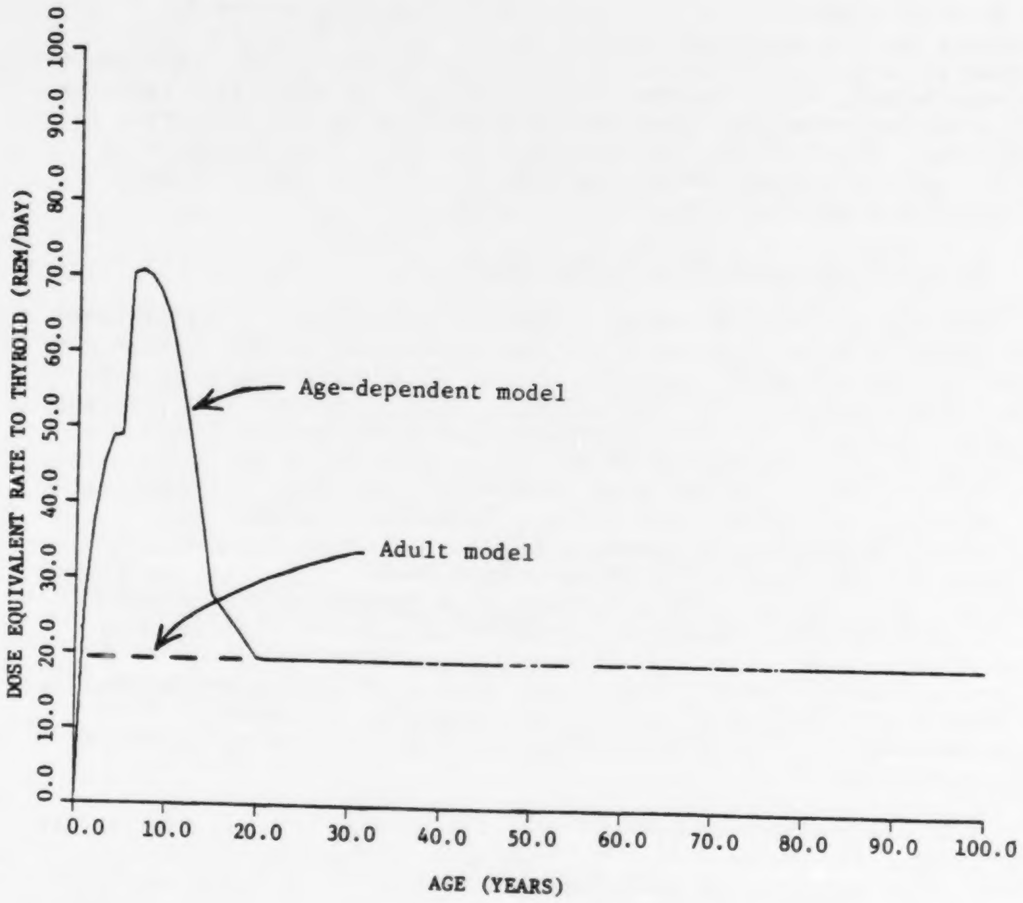

Figure 7.5-2. Dose from chronic inhalation of iodine-131 in air at a concentration of 1 μCi/m^3.

Two subcompartments, surface and volume, are considered within each of these main compartments. The four subcompartments of mineralized skeleton and the movement of strontium among these compartments are shown schematically in Figure 7.5-3. The equations governing the age dependence of the parameters are given in (ORNL84a). Dose rate curves for the inhalation and ingestion of constant concentrations of strontium-90 are given in Figures 7.5-4 and 7.5-5. The comparable curves for Reference Man are again indicated by dashed lines. Thus, for this element and organ combination, the dose rate resulting from ingestion is somewhat higher, while the dose rate resulting from inhalation exhibits only minor perturbations, when the age dependence of the parameters is considered. The lifetime (70-year) dose resulting from ingestion is about 7 percent greater and the inhalation dose less than 1 percent different when age dependence is considered.

Plutonium and Lung and Red Bone Marrow

Apparently plutonium and iron bear sufficient chemical resemblance that plutonium is able to penetrate some iron transport and storage systems. It has been shown that plutonium in blood serum complexes with transferrin, the iron-transport protein. Thus, plutonium will partially trace the iron pathway, with the result that a substantial fraction of systemic plutonium is carried to the bone marrow and to the liver. In the skeleton, plutonium may be released mainly at sites of developing red cells. Plutonium that has reached the skeleton behaves very differently from iron; its movement is governed by fairly complicated processes of bone resorption and addition. Because the total metabolic behavior of plutonium is not closely related to that of any essential element, any retention model for plutonium as a function of age will involve much larger uncertainties than the analogous model for strontium. Still, there is enough information concerning the metabolism of plutonium by mammals to justify an examination of potential differences with age in doses to radiosensitive tissues following intake of this radioelement.

The effect of age-dependent parameters on dose rate calculations is most evident for the lung when the inhalation pathway is considered. Figure 7.5-6 exhibits the variation in dose rate to the total and pulmonary portions of the lung both for the adult and age-dependent cases. The increased dose rate from age 0 to about 20 is typically caused by variations in the breathing rate-lung mass ratio for infants and juveniles. For this model, the age-dependent pulmonary lung 70-year dose is about 9 percent greater than for the adult model.

To describe retention of plutonium in the skeleton, it is convenient to view the skeleton as consisting of a cortical compartment and trabecular compartment. Each of these is further divided into three subcompartments: bone surface, bone volume, and a transfer compartment. The transfer compartment, which includes the bone marrow, may receive plutonium that is removed from bone surface or volume; plutonium may reside in this compartment temporarily before being returned either to

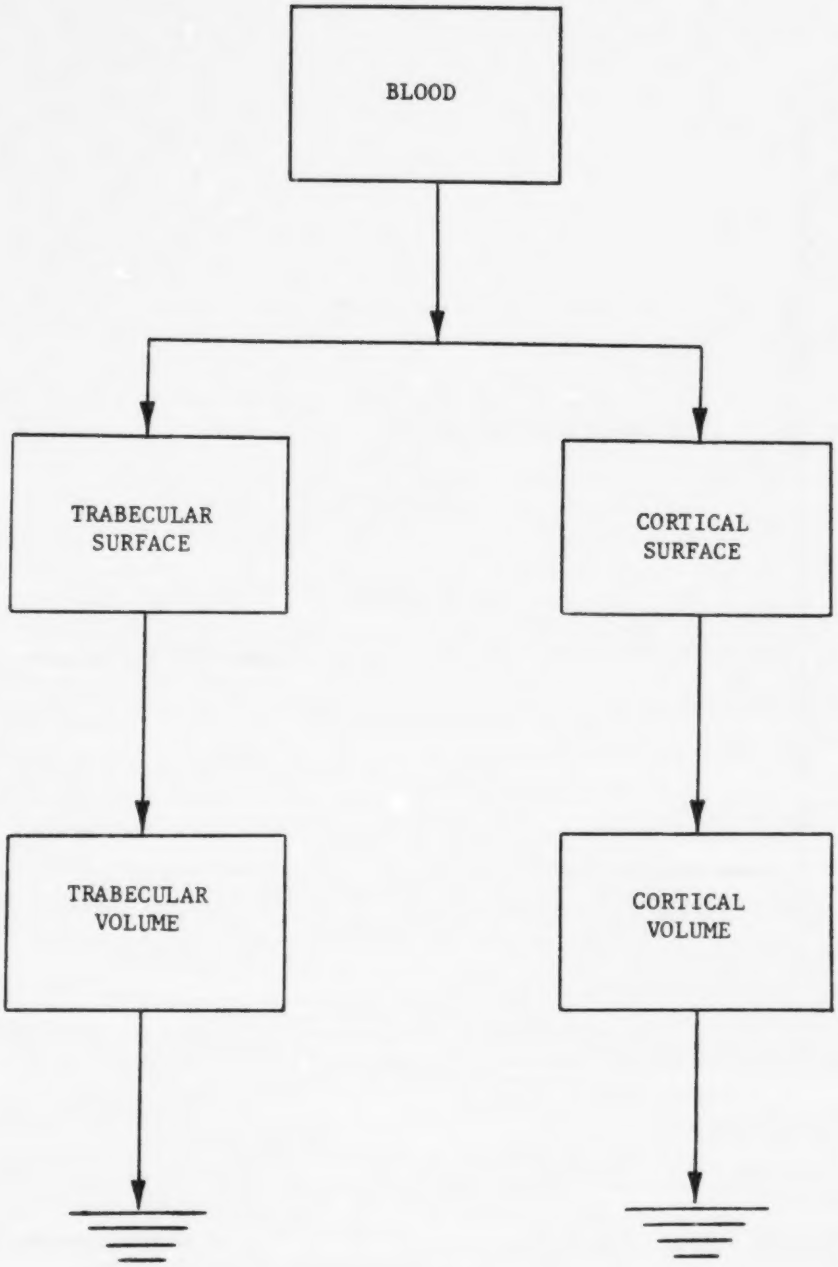
BLOOD
TRABECULAR SURFACE
CORTICAL SURFACE
TRABECULAR VOLUME
CORTICAL VOLUME

Figure 7.5-3. Compartments and pathways in model for strontium in skeleton.

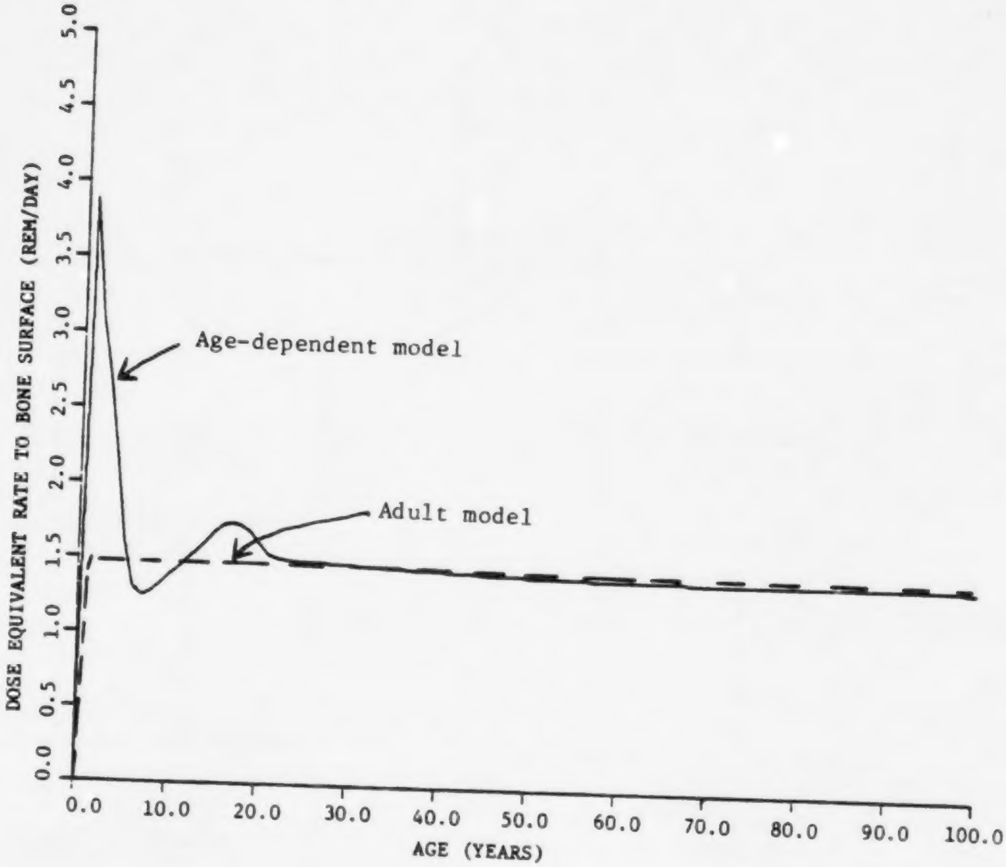

Figure 7.5-4. Dose from chronic ingestion of strontium-90 in in water at a concentration of 1 μCi/l.

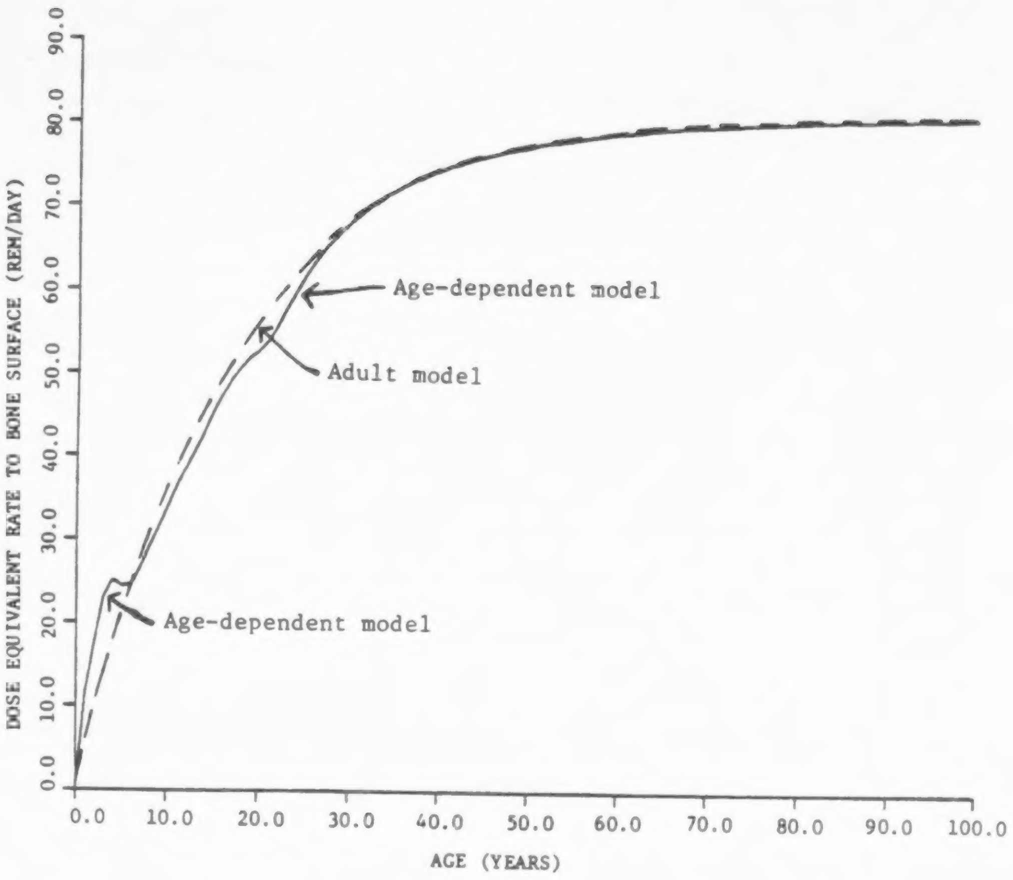

Figure 7.5-5. Dose from chronic inhalation of strontium-90 in air at a concentration of 1 μCi/m^3.

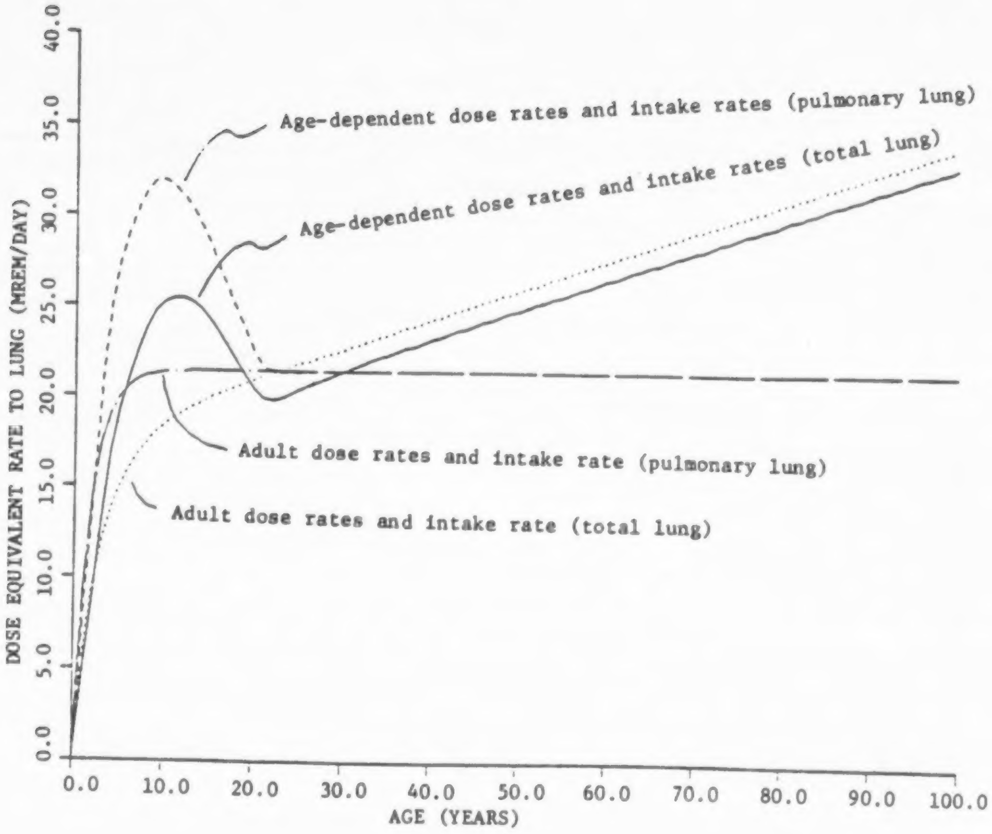

Figure 7.5-6 Dose from chronic inhalation of pultonium-239 in air at a concentration of 1 $\mu Ci/m^3$.

the bloodstream or to bone surfaces (Figure 7.5-7). Because of the large amount of recycling of plutonium among the skeletal compartments, blood, and other organs, recycling is considered explicitly in the model. The age-dependent features of the model are described in detail in (ORNL84a).

Red bone marrow dose rates for the age dependent model are shown in Figure 7.5-8, for ingestion, and in Figure 7.5-9, for inhalation. The dashed curves are the dose rates using non-age-dependent parameters. As in the corresponding curves for strontium, the difference is more pronounced for the ingestion pathway. Because of the long radiological and biological half-lives of plutonium in the skeleton, the dose rate, for a chronic intake, does not reach equilibrium within the one hundred year time period of the figures. The total lifetime (70-year) dose to the red marrow is about 25 percent greater for ingestion, and nearly unchanged for inhalation when the age-dependent parameters are used.

In summary, it is difficult to make generalizations concerning the uncertainty involved in neglecting age dependence in the dose calculations. Although the examples given indicate higher dose rates for the ingestion pathway, with smaller changes for inhalation, when using age-dependent parameters, this results from the complex interaction between parameters in the dose equation and depends on the element/organ combination under consideration.

7.5.3 Dose Uncertainty Caused by Measurement Errors

The last potential source of uncertainty in the dose calculations is the error involved in making measurements of fixed quantities (ORNL84b). The radioactive half-life of an isotope, for example, may be measured independently of any biological system, but the measurement is subject to some error. The organ mass of a given organ may also be measured with only a small error. Repeated determinations of these quantities, in addition, can reduce the error. Although this source of uncertainty may be of importance in other aspects of an environmental assessment, it is of little consequence in the dosimetry, because it is overwhelmed by the magnitude of the uncertainties resulting from age and individual variations.

Although consideration of the factors described above implies large uncertainties in calculated doses, the actual variation is expected to be considerably smaller. The reason for this, and some supporting studies on real populations, are presented in Section 7.6.

7.6 Distribution of Doses in the General Population

Although the use of extreme parameter values in a sensitivity analysis indicates that large uncertainties in calculated doses are possible, this uncertainty is not usually reflected in the general population. There are several reasons for this: the parameter values chosen are intended to be typical of an individual in the population; it is improbable that the "worst case" parameters would be chosen for all

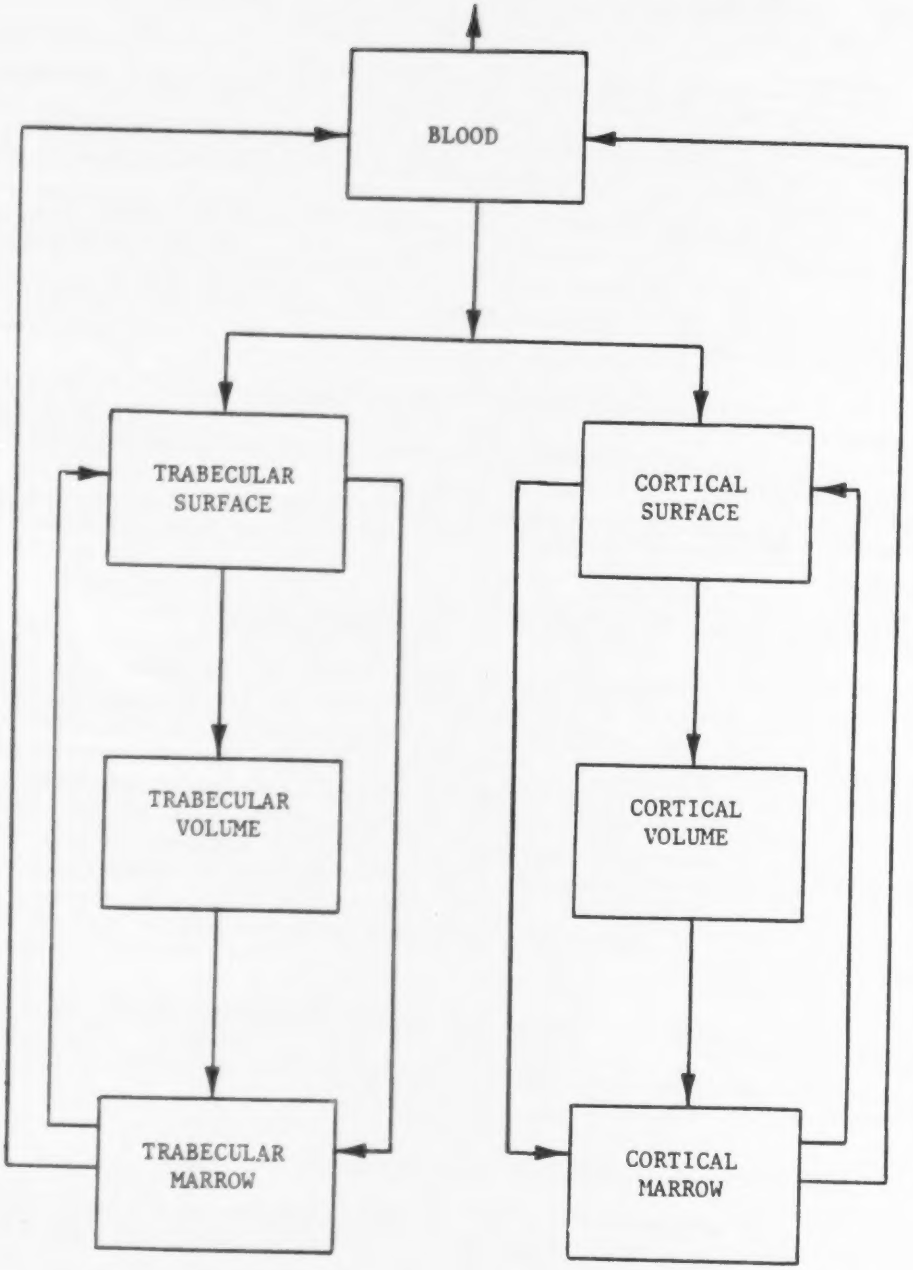
BLOOD
TRABECULAR SURFACE
CORTICAL SURFACE
TRABECULAR VOLUME
CORTICAL VOLUME
TRABECULAR MARROW
CORTICAL MARROW

Figure 7.5-7. Compartments and pathways in model for plutonium in skeleton.

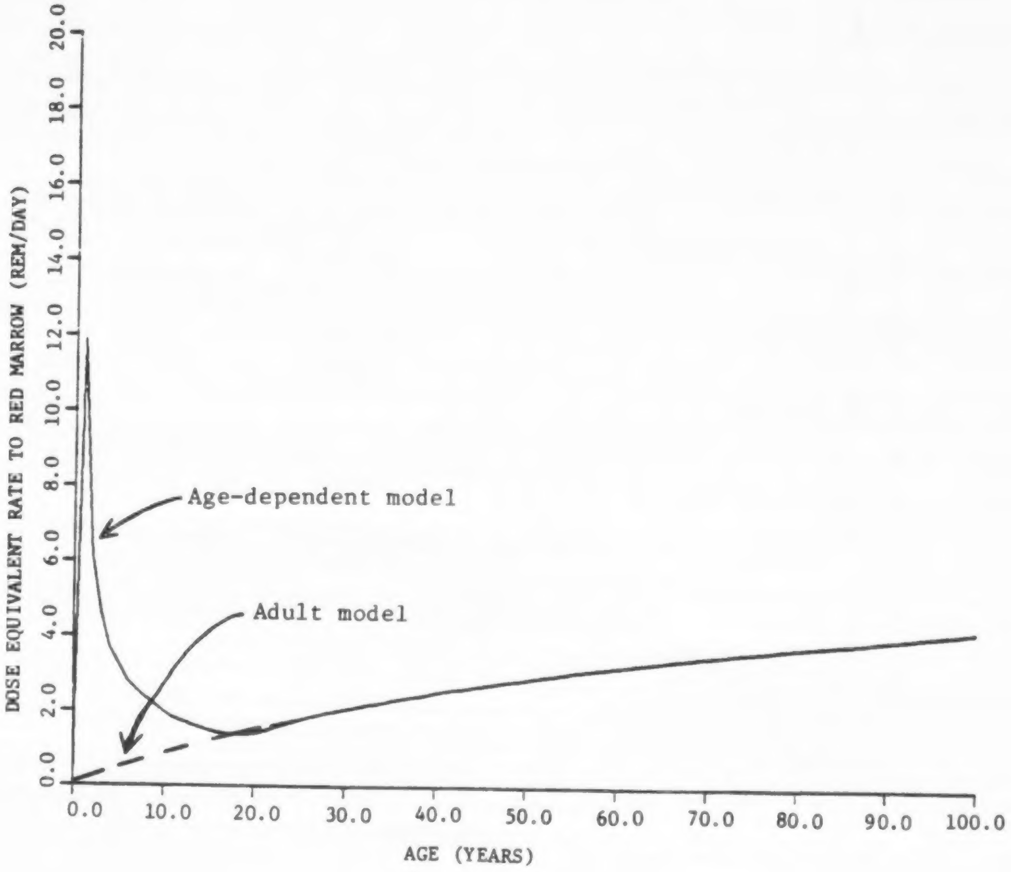

Figure 7.5-8. Dose from chronic ingestion of plutonium-239 in water at a concentration of 1 μCi/l.

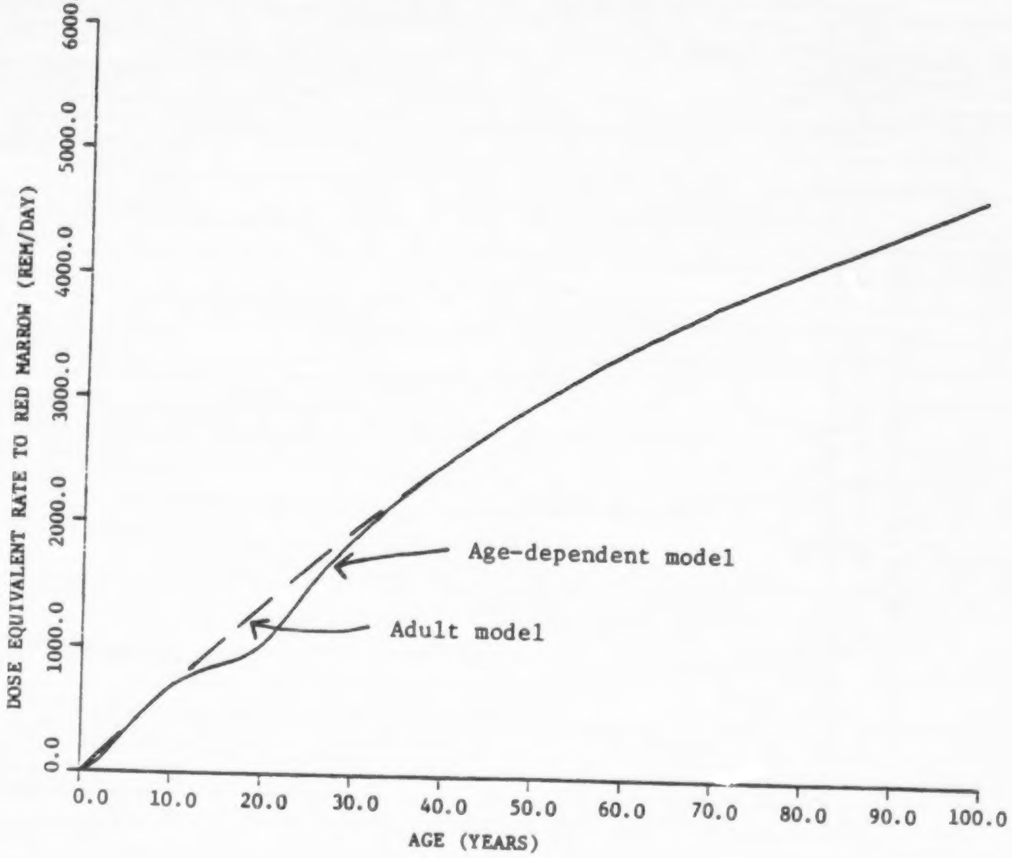

Figure 7.5-9. Dose from chronic inhalation of plutonium-239 in air at a concentration of 1 $\mu Ci/m^3$.

terms in the equation; and not all of the terms are mutually independent, e.g., an increased intake may be offset by more rapid excretion.

This smaller range of uncertainty in real populations is demonstrated by studies performed on various human and animal populations. It should be noted that there is always some variability in observed doses that results primarily from differences in the characteristics of individuals. The usual way of specifying the dose, or activity, variability in an organ is in terms of the deviation from the average, or mean, value. In the following studies, it should also be noted that, in addition to the variability resulting from individual characteristics, the exposure levels of individuals may also have varied appreciably - another factor tending to increase the dose uncertainty. The following studies are representative of those carried out on real populations:

(1) An analysis of the thyroid from 133 jackrabbits in a nuclear fallout area (Tu65) found that in only 2 did the iodine-131 content exceed three times the sample mean.

(2) Measurements of the strontium-90 content of adult whole skeletons (Ku62) showed that only about 5 percent of the population would exceed twice the average activity, with only about 0.1 percent exceeding four times the average.

(3) In another study, the cesium-137 content of 878 skeletal muscle samples (E164a, b) was measured. This radioisotope is also the result of nuclear tests so that the muscle content depends not only on the variation in individual parameters but also on the pathways leading to ingestion or inhalation of the isotope. Nevertheless, analyses of these samples indicated that only 0.2 percent exceeded three times the mean activity at a 95 percent confidence level.

(4) A study of the variability in organ deposition among individuals exposed under relatively similar conditions to toxic substances has also been performed (Cu79). In eleven exposure situations (Table 7.6-1), the geometric standard deviation of the apparently lognormal organ doses ranged from 1.3 to 3.4. This means that about 68 percent of the organ doses were between 1/6 times and 6 times the geometric mean of the doses. From the table, for example, 68 percent of the bone doses resulting from ingestion of strontium-90 would lie between 0.56 and 1.8 times the average.

In all but two of the situations examined, there is the complicating factor that there was probably a great deal of variation in the exposure levels experienced by members of the population. The magnitude of geometric standard deviations of the studies listed in Table 7.6-1 may be the evidence of this variation since, except for the two beagle studies, the exposure was not uniform. Despite these nonuniform exposures, however, the organ dose is not greatly affected probably because of differences in metabolic processes. For example, there is probably some "self-adjustment" in the amount of strontium-90 absorbed

Table 7.6-1. Distributions of organ doses[a] from inhalation and ingestion of metals

Population	Exposure	Principal exposure mode	Target organ	Geometric standard deviation of organ doses[a]
Beagle	Metals	Inhalation	Bone or liver	1.8
Humans	Plutonium (fallout)	Inhalation	Lung	3.1[b]
Humans	Titanium (soil)	Inhalation	Lung	3.4[b]
Humans	Aluminum (soil)	Inhalation	Lung	3.4[b]
Humans	Vanadium (fuel combustion)	Inhalation	Lung	3.4[b]
Beagles	Strontium-90	Ingestion	Bone	1.3
Humans	Strontium-90 (fallout)	Ingestion	Bone	1.8[b]
Humans (smokers)	Cadmium	Inhalation and Ingestion	Kidney	1.8[b]
Humans (nonsmokers)	Cadmium	Inhalation and Ingestion	Kidney	1.8[b]
Humans	Lead	Inhalation and Ingestion	Bone	2.2[b]
Humans	Lead	Inhalation	Lung	1.7[b]

(a)The stable element organ doses used in compiling this table were generally expressed in parts-per-million of organ mass.

(b)Note that exposure levels may vary considerably among individuals; if this factor could be eliminated, geometric standard deviations probably would be smaller.

Source: (Cu79).

from the small intestine to blood of different persons, since strontium-90 tends to vary with calcium in food; if a person has a low calcium intake, then he may absorb a higher fraction of the calcium and strontium-90 than a person with a high calcium intake.

In the beagle studies, the geometric standard deviation is 1.8 for inhaled metals in bone or liver, but is only 1.3 for ingested strontium-90 in bone. An important difference is that all dogs ingesting strontium-90 at a given level were administered the same amount, whereas, in the inhalation studies, the exposure air concentrations were controlled but the dogs inhaled variable amounts depending upon their individual characteristic breathing patterns.

Thus, in real situations, the overall uncertainty in dose is seen to be considerably smaller than would be expected solely on a basis of the "worst case" sensitivity analyses.

7.7 Summary

This chapter presents an overview of the methods used by EPA to estimate radiation doses. The chapter defines the basic quantities reported by EPA and describes briefly the models employed. The chapter also points out departures from the occupational parameters and assumptions employed in the basic ICRP methodology and gives the reasons for the deviations outlined.

Many of the physiological and metabolic parameters recommended in methods for calculating radiation doses are based on a limited number of observations, often on atypical humans or on other species. EPA has attempted to bound the uncertainty associated with the ranges observed for some of the more important parameters used. In fact, some empirical data on population doses mentioned here indicate that actual dose uncertainties are much less than is implied by this "worst case" analysis. For the sources of uncertainty discussed, the large dose ranges possible because of variation in individual characteristics must be modified by consideration of the narrower ranges indicated by studies of real populations; the dose range resulting from age dependence appears to be small for lifetime exposures, and the range resulting from experimental error is negligible by comparison. Based on these observations, it is reasonable to estimate that EPA's calculated doses should be accurate within a factor of three or four. It should be emphasized that much of the "uncertainty" in the dose calculation is not caused by parameter error but reflects real differences in individual characteristics within the general population. Therefore, the uncertainty in the dose estimates cannot be dissociated from specification of the segment of the population to be protected.

More complete derivations and explanations for the EPA methodology are given in the references cited in the text, and a technical description of the dose rate equations and their use in conjunction with the life table risk evaluation is given in Addendum B.

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Chapter 8: ESTIMATING THE RISK OF HEALTH EFFECTS RESULTING FROM RADIONUCLIDE AIR EMISSIONS

8.1 Introduction

This chapter describes how the Environmental Protection Agency (EPA) estimates the probability of fatal cancer, serious genetic effects, and other detrimental health effects resulting from exposure to ionizing radiation. Such risk estimates are complex. They are also uncertain, even though much scientific effort has been expended to increase the understanding of radiation effects.

Because the effects of radiation on human health are known more quantitatively than are the effects of most other environmental pollutants, it is possible to make numerical estimates of the risk that may occur as a result of a particular source of radioactive emissions. Such numbers may give an unwarranted aura of certainty to estimated radiation risks. Compared to the baseline incidence of cancer and genetic defects, radiogenic cancer and radiation-induced genetic defects do not occur very frequently. Even among heavily irradiated populations, the number of cancers and genetic defects resulting from radiation is not known with either accuracy or precision simply because of sampling variability. In addition, exposed populations have not been followed for their full lifetime, so that information on ultimate effects is limited. Moreover, when considered in light of information gained from experiments with animals and from various theories of carcinogenesis and mutagenesis, the observational data on the effects of human exposure are subject to a number of interpretations. This in turn leads to differing estimates of radiation risks by both individual radiation scientists and expert groups. Readers should bear in mind that estimating radiation risks is not a mature science and that the evaluation of radiation hazards will change as additional information becomes available. In this chapter a number of simple mathematical models are presented that may describe the main features of the human response to radiation. However, most scientists would agree that the underlying reality is quite complicated and largely unknown, so that such models should not be taken too literally but rather as useful approximations that will some day be obsolete.

The risk estimates in the Draft Background Information Document (DBID) (EPA83a) for the proposed rules on radionuclide emissions were based on the 1972 National Academy of Science BEIR report (NAS72). To take advantage of more recent data and analysis of radiation risks, EPA's estimates of cancer and genetic risks in this final BID are based on the BEIR-3 report (NAS80). This report was prepared for the purpose of assessing radiation risks at the low exposure levels of interest in standard setting. As phrased by the President of the Academy, "We believe that the report will be helpful to the EPA and other agencies as they reassess radiation protection standards. It provides the scientific bases upon which standards may be decided after nonscientific social values have been taken into account."

In this chapter, we outline the various assumptions made in calculating radiation risks based on the 1980 NAS report and compare these risk estimates with those prepared by other scientific groups such as the 1972 NAS BEIR Committee (NAS72), the United Nations Scientific Committee on the Effects of Atomic Radiation (UNSCEAR), and the International Commission on Radiation Protection (ICRP). We recognize that information on radiation risks is incomplete and do not argue that the estimates made by the 1980 NAS BEIR Committee are highly accurate. Rather, we discuss some of the deficiencies in the available data base and point out possible sources of bias in current risk estimates. Nevertheless, we do believe the risk estimates made by EPA are "state-of-the-art".

The analysis of possible health effects resulting from radionuclide emissions in the air, EPA83a, indicated that by far the greatest risk was radiogenic cancer, primarily lung cancer caused by inhaling radioactive material. The risk of genetic damage was typically 10 to 100 times smaller than the risk of radiogenic cancer. Although we include a discussion of possible genetic effects and other health hazards due to radiation in this chapter, EPA has not included estimates of genetic damage for the sources of radionuclide emissions described in Chapters 11-17 of Volume II of the BID. As outlined in Section 8.7 below, the additional risk of genetic harm is so much smaller than the uncertainty in the estimated risk of radiogenic cancer, that it has not been a factor in this rulemaking.

In the sections below, we first consider the cancer risk resulting from whole-body exposure to low-LET* radiation, i.e., lightly ionizing radiation like the energetic electrons produced by X-rays or gamma rays. Environmental contamination by radioactive materials also leads to the ingestion or inhalation of the material and subsequent concentration of the radionuclides in selected body organs. Therefore, the cancer risk resulting from low-LET irradiation of specific organs is examined next. Organ doses can also result from high-LET radiation, such as that associated with alpha particles. Estimation of cancer risks for situations where high-LET radiation is distributed more or less uniformly within a body organ is the third situation considered, Section 8.3. Because highly ionizing alpha particles have a very short range in tissue, there are exposure situations where the dose distribution to particular organs is extremely nonuniform. An example is inhaled radon progeny: polonium-218, lead-214, and polonium-214. For these radionuclides we base our cancer risk estimates on the amount of radon progeny inhaled rather than on the estimated dose, which is highly nonuniform and cannot be well quantified. Therefore, risk estimates of radon exposure are examined separately in Section 8.4. We review the causes of uncertainty in the cancer risk estimates and the magnitude of this uncertainty in Section 8.5, so that the public as well as EPA decision makers have a proper understanding of the degree of confidence to place in them. In Section 8.6, we review and quantify the hazard of deleterious genetic effects due to radiation and the effects of exposure in utero on the developing fetus. Finally, in section 8.7, we calculate cancer and genetic risks from background radiation using the models described in this chapter.

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* Linear Energy Transfer (LET), the energy deposited per unit of distance along the path of a charged particle.

8.2 Cancer Risk Estimates for Low-LET Radiations

Most of the observations of radiation-induced carcinogenesis in humans are on groups exposed to low-LET radiations. These groups include the Japanese A-bomb survivors and medical patients treated with X-rays for ankylosing spondylitis in England from 1935 to 1954 (Sm78). The UNSCEAR (UNSCEAR77) and NAS Committee on the Biological Effects of Ionizing Radiations (BEIR) (NAS80) have provided knowledgeable reviews of these and other data on the carcinogenic effects of human exposures.

The most important epidemiological data base on radiogenic cancer is the A-bomb survivors. The Japanese A-bomb survivors have been studied for more than 38 years and most of them, the Life Span Study Sample, have been followed in a carefully planned and monitored epidemiological survey since 1950 (Kab82, Wa83). They were exposed to a wide range of doses and are the largest group that has been studied. Therefore, they are virtually the only group providing information on the response pattern at various levels of exposure to low-LET radiation. Unfortunately, the doses received by various individuals in the Life Span Study Sample are not yet accurately known. The 1980 BEIR Committee's analysis of the A-bomb survivor data was prepared before bias in the dose estimates for the A-bomb survivors (the tentative 1965 dose estimates, T65) became widely recognized (Lo81). It is now clear that the T65 doses tended to be overestimated (Bo82, RERF83,84) so that the BEIR Committee's estimates of the risk per unit dose are likely to be too low. A detailed reevaluation of current risk estimates is indicated when the A-bomb survivor data have been reanalyzed on the basis of new and better estimates of the dose to individual survivors.

Uncertainties in radiation risk estimates do not result just from the uncertainties in the Japanese data base and in other epidemiological studies. Analyses of these data bases require a number of assumptions that have a considerable effect on the estimated risk. These assumptions are discussed below. The degree of uncertainty introduced by choosing among these assumptions is probably greater than the uncertainty of the estimated risk per unit dose among the A-bomb survivors or other sources of risk estimates for radiogenic cancer in humans.

8.2.1 Assumptions Needed to Make Risk Estimates

A number of assumptions must be made about how observations at high doses should be applied at low doses and low dose rates for radiation of a given type (LET). These assumptions include the shape of the dose response function and possible dose rate effects. A dose response function expresses the relationship between dose and the probability that a radiogenic cancer is induced. Observed excess cancers have occurred, for the most part, following relatively high doses of ionizing radiation compared to those likely to occur as a result of the combination of background radiation and environmental contamination from controllable sources of radiation. Therefore, a dose response model provides a method of interpolating between the number of radiogenic cancers observed at high doses and the number of cancers resulting from all causes including background radiation.

The range of interpolation is not the same for all kinds of cancer because it depends upon the radiosensitivity of a given tissue. For example, the most probable radiogenic cancer for women is breast cancer. As described below, with appropriate references, breast cancer appears not to be reduced when the dose is delivered over a long period of time. For example, the number of excess cancers per unit dose among Japanese women, who received acute doses, is about the same per unit dose as women exposed to small periodic doses of X-rays over many years. If this is actually the case, background radiation is as carcinogenic for breast tissue as the acute exposures from A-bomb gamma radiation. Moreover, the female A-bomb survivors show an excess of breast cancer at doses below 20 rad which is linearly proportional to that observed at several hundred rad (Tob84). Women in their forties, the youngest age group in which breast cancer is common, have received about 4 rad of whole-body low-LET background radiation and usually some additional dose incurred for diagnostic medical purposes. Therefore, for this cancer, the difference between observed radiogenic cancer, less than 20 rad, and the dose resulting from background radiation is less than a factor of five, not several orders of magnitude as is sometimes claimed. However, it should be noted that breast tissue is a comparatively sensitive tissue for cancer induction and that for most cancers, a statistically significant excess has not been observed at doses below 100 rad, low LET. Therefore, the range of dose interpolation between observed and calculated risk is often large.

8.2.2 Dose Response Functions

The 1980 NAS report (NAS80) examined three dose response functions in detail: (1) linear, in which effects are directly proportional to dose at all doses; (2) linear quadratic, in which effects are very nearly proportional to dose at very low doses and proportional to the square of the dose at high doses; and (3) a quadratic dose response function, where the risk varies as the square of the dose at all dose levels.

We believe the first two of these functions are compatible with most of the data on human cancer. Information that became available only after the BEIR-3 report was published indicates that a quadratic response function is inconsistent with the observed excess risk of solid cancers at Nagasaki, where the estimated gamma-ray doses are not seriously confounded by an assumed neutron dose component. The chance that a quadratic response function underlies the excess cancer observed in the Nagasaki incidence data has been reported as only one in ten thousand (Wa83). Although a quadratic response function is not incompatible with the Life Span Study Sample data on leukemia incidence at Nagasaki, Beebe and others (Be78, Ela77) have pointed out how unrepresentative these data are of the total observed dose response for leukemia in that city. There is no evidence that a quadratic response function provides a better fit to the observed leukemia excess among all A-bomb survivors in the Life Span Study Sample than a simple linear model (NAS80). Based on these considerations, we do not believe a quadratic response can be used in a serious effort to estimate cancer risks due to ionizing radiation. EPA notes that neither the NCRP, the ICRP, nor other authorative scientific groups, e.g., NRPB and UNSCEAR, have used a quadratic response function to estimate the risks due to ionizing radiation.

The 1980 NAS BEIR Committee considered only the Japanese mortality data in their analysis of possible dose response functions (NAS80). Based on the T65 dose estimates, this Committee showed that the excess incidence of solid cancer and leukemia among the A-bomb survivors is compatible with either a linear or linear quadratic dose response to the low-LET radiation component and a linear response to the high-LET neutron component (NAS80). Although the 1980 BEIR report indicated that low-LET risk estimates based on a linear quadratic response were "preferred" by most of the scientists who prepared that report, opinion was not unanimous, and we believe the subsequent reassessment of the A-bomb dose seriously weakens the Committee's conclusion. The Committee's analysis of dose response functions was based on the assumption that most of the observed excess leukemia and solid cancers among A-bomb survivors resulted from neutrons (NAS80). Current evidence, however, is conclusive that neutrons were only a minor component of the dose in both Hiroshima and Nagasaki (Bo82, RERF83,84). Therefore, it is likely that the linear response attributed to neutrons was caused by the gamma dose, not the dose from neutrons. This point is discussed further in Section 8.5.

Reanalysis of the Japanese experience after completion of the dose reassessment may provide more definitive information on the dose response of the A-bomb survivors, but it is unlikely to provide a consensus on the dose response at environmental levels, i.e., about 100 mrad per year. This is because at low enough doses there will always be sampling variations in the observed risks so that observations are compatible, in a statistical sense, with a variety of dose response functions. In the absence of empirical evidence or a strong theoretical basis, a choice between dose response functions must be based on other considerations.

Although there is evidence for a nonlinear response to low-LET radiations in some, but not all, studies of animal radiocarcinogenesis (see below), we are not aware of any similar data on human cancers that are incompatible with a simple linear model. In such a case, it may be preferable to adopt the simplest hypothesis that adequately models the observed radiation effect. Occams's razor is still a viable scientific rule for separating necessary from _ad hoc_ assumptions. Moreover, EPA believes that risk estimates for the purpose of assessing radiation impacts on public health should be based on scientifically creditable risk models that are unlikely to understate the risk. The linear model fulfills this criterion. Given the current bias in the doses assigned to A-bomb survivors (see 8.5.1), such an approach seems reasonable, as well as prudent. Therefore, EPA has utilized the BEIR-3 _linear dose response_ model as one of two dose response models for estimating the risk of radiogenic cancer due to low-LET radiations.

For low-LET radiations, we have also included risk estimates that are based on the BEIR-3 _linear quadratic dose response_ model. While in the dose range of interest (environmental levels) the dose squared term in this model is insignificant, the linear term is about 2.5 times smaller than that in the BEIR-3 linear response model, NAS80. That is, for the same dose, risk estimates based on the BEIR-3 linear quadratic dose response model are only 40 percent of those based on the BEIR-3 linear model.

Many of the risk estimates needed to evaluate the effect of radionuclide emission must be made on an organ-specific basis. The BEIR-3 report provides risk coefficients for individual solid cancers only for the linear model, Tables V-14 and V-15 in NAS80. We have therefore divided BEIR-3 risk organ estimates for a linear response by a factor of 2.5 to obtain organ-specific linear quadratic risk coefficients.

The underlying basis for a linear quadratic response is thought to be that repair of radiation damage mitigates the effect of small doses of radiation or those which occur over a long time period, the reduced linear term being indicative of this repair. Use of a linear quadratic dose response function, as formulated by the BEIR-3 committee, is equivalent to the use of a dose rate effectiveness factor (DREF) of 2.5 (see below).

8.2.3 The Possible Effects of Dose Rate on Radiocarcinogenesis

The BEIR-3 committee limited its risk estimates to a minimum dose rate of 1 rem per year and stated that it "does not know if dose rates of gamma rays and X-rays of about 100 mrad/y are detrimental to man." At dose rates comparable to the annual dose that everyone receives from naturally occurring radioactive materials, a considerable body of scientific opinion holds that the effects of radiation are reduced. NCRP Committee 40 has suggested that carcinogenic effects of low-LET radiations may be a factor of from 2 to 10 times less for small doses and dose rates than have been observed at high doses (NCRP80).

The low dose and low dose rate effectiveness factors developed by NCRP Committee 40 are based on their analysis of a large body of plant and animal data that showed reduced effects at low doses for a number of biological endpoints, including radiogenic cancer in animals, chiefly rodents. However, no data for cancer in humans confirm these findings as yet. A few human studies contradict them. Highly fractionated small doses to human breast tissue are apparently as carcinogenic as large acute doses (NAS80, Laa80). Furthermore, small acute (less then 10 rad) doses to the thyroid are as effective per rad as much larger doses in initiating thyroid cancer (UNSCEAR77, NAS80). Moreover, the increased breast cancer resulting from chronic low-dose occupational gamma-ray exposures among British dial painters is comparable to, or larger, than that expected on the basis of acute high-dose exposures (Ba81).

While none of these examples is persuasive by itself, collectively they indicate that it may not be prudent to assume that all kinds of cancer are reduced at low dose rates and/or low doses. However, it may be overly conservative to estimate the risk of all cancers on the basis of the linearity observed for breast and thyroid cancer. The International Commission on Radiation Protection and the United Nations Scientific Committee on Atomic Radiations have used a dose rate effectiveness factor of about 2.5 to estimate the risks from occupational (ICRP77) and environmental exposures (UNSCEAR77). Their choice of a DREF is fully consistent with and equivalent to the reduction of risk at low doses obtained by substituting the BEIR-3 linear-quadratic response model for their linear model. Use of both a DREF and a linear quadratic model for risk estimation is inappropriate (NCRP80).

The difference between risk estimates obtained with the BEIR-3 linear and linear-quadratic dose response models is by no means the full measure of the uncertainty in the estimates of the cancer risk resulting from ionizing radiation (Section 8.5 summarizes information on uncertainty). The use of two dose models serves as a reminder that there is more than one creditable response model for estimating radiation risks and that it is not known if all radiogenic cancers have the same dose response.

8.2.4 Risk Projection Models

None of the exposed groups has been observed long enough to assess the full effects of their exposures, if, as is currently thought, most radiogenic cancers occur throughout an exposed person's lifetime (NAS80). Therefore, another major choice that must be made in assessing the lifetime cancer risk resulting from radiation is to select a risk projection model to estimate the risk for a longer period of time than currently available observation data will allow.

To estimate the risk of radiation exposure that is beyond the years of observation, either a relative risk or an absolute risk projection model (or suitable variations) must be used. These models are described at length in Chapter 4 of the 1980 NAS report (NAS80). The relative risk projection model projects the currently observed percentage increase in cancer risk per unit dose into future years. An absolute risk model projects the average observed number of excess cancers per unit dose into the future years at risk.

Because the underlying risk of cancer increases rapidly with age, the relative risk model predicts a larger probability of excess cancer toward the end of a person's lifetime. In contrast, the absolute risk model predicts a constant incidence of excess cancer across time. Therefore, given the incomplete data we have now, less than lifetime follow-up, a relative risk model projects somewhat greater risk than that estimated using an absolute risk model.

The National Academy of Sciences BEIR Committee and other scientific groups, e.g. UNSCEAR, have not concluded which projection model is the appropriate choice for most radiogenic cancers. However, evidence is accumulating that favors the relative risk projection model for most solid cancers. As pointed out by the 1980 NAS BEIR Committee,

> "If the relative-risk model applies, then the age of the exposed groups, both at the time of exposure and as they move through life, becomes very important. There is now considerable evidence in nearly all the adult human populations studied that persons irradiated at higher ages have, in general, a greater excess risk of cancer than those irradiated at lower ages, or at least they develop cancer sooner. Furthermore, if they are irradiated at a particular age, the excess risk tends to rise *pari passu* [at equal pace] with the risk of the population at large. In other words, the relative-risk model with respect to cancer susceptibility at least as a function of age, evidently applies to some kinds of cancer that have been observed to result from radiation exposure." (NAS80, p. 33)

This observation is confirmed by the Ninth A-bomb Survivor Life Span Study, published 2 years after the 1980 Academy report. This latest report indicates that, for solid cancers, relative risks have continued to remain constant in recent years while absolute risks have increased substantially (Kab82). Smith and Doll (Sm78) have reached similar conclusions on the trend in excess cancer with time among the irradiated spondylitic patients.

Although we believe considerable weight should be given to the relative risk model for most solid cancers (see below), the model does not necessarily give an accurate projection of lifetime risk. The mix of tumor types varies with age so that the relative frequency of some common radiogenic tumors, such as thyroid cancer, decreases for older ages. Land has pointed out that this may result in overestimates of the lifetime risk when the estimates are based on a projection model using relative risks (Lac83). While this may turn out to be true for estimates of cancer incidence that include cancers less likely to be fatal, e.g., thyroid, it may not be too important in estimating the lifetime risk of fatal cancers, since the incidence of most of the common fatal cancers, e.g., breast and lung cancers, increases with age.

Leukemia and bone cancer are exceptions to the general validity of a lifetime expression period for radiogenic cancers. Most, if not all, of the leukemia risk has apparently already been expressed in both the A-bomb survivors and the spondylitics (Kab82, Sm78). Similarly, bone sarcoma from acute exposure appears to have a limited expression period (NAS80, Mab83). For these diseases, the BEIR-3 Committee believed that an absolute risk projection model with a limited expression period is appropriate for estimating lifetime risk (NAS80).

It should be noted that unlike the NAS BEIR-1 report (NAS72) the BEIR-3 Committee's relative and absolute risk models are age dependent. That is, the risk coefficient changes depending on the age of the exposed persons. Observation data on how cancer risk resulting from radiation changes with age are sparse, particularly so in the case of childhood exposures. Nevertheless, the explicit consideration of the variation in radiosensitivity with age at exposure is a significant improvement in methodology. It is important to differentiate between age sensitivity at exposure and the age dependence of cancer expression. In general, people are most sensitive to radiation when they are young. In contrast, most radiogenic cancers occur late in life, much like cancers resulting from other causes. In this chapter we present risk estimates for a lifetime exposure of equal annual doses. The cancer risk estimated is lifetime risk from this exposure pattern. However, age dependent analyses using BEIR-3 risk coefficients indicate that the risk from one year of exposure varies by a factor of at least five depending on the age of the recipient.

8.2.5 Effect of Various Assumptions on the Numerical Risk Estimates

Differences between risk estimates made by using various combinations of the assumptions described above were examined in the 1980 NAS report. Table 8.2-1, taken from Table V-25 (NAS80), shows the range of cancer fatalities induced by a single 10-rad dose as estimated using linear, linear quadratic, and quadratic dose response functions and two projection models, relative and absolute risk.

As illustrated in Table 8.2-1, estimating the cancer risk for a given projection model on the basis of a quadratic as compared to a linear dose response reduces the estimated risk of fatal cancer by a factor of nearly 20. Between the more credible linear and linear quadratic response functions the difference is less, a factor of about two and a half. For a given dose response model, results obtained with the two projection models, for solid cancers, differ by about a factor of three.

Even though the 1980 NAS analysis estimated lower risks for a linear quadratic response, it should not be concluded that this response

Table 8.2-1. Range of cancer fatalities induced by 10 rad low-LET radiation (Average value per rad per million persons exposed)

Dose Response Functions	Lifetime Risk Projection Model	
	Relative[(a)]	Absolute
Linear[(b)]	501	167
Linear Quadratic[(c)]	226	77
Quadratic[(d)]	28	10

(a) Relative risk projection for all solid cancers except leukemia and bone cancer fatalities, which are projected by means of the absolute risk model (NAS80).

(b) Response R varies as a constant times the dose, i.e., $R=C_1D$.

(c) $R=C_2D+C_3D^2$.

(d) $R=C_4D^2$.

Source: NAS80, Table V-25.

function always provides smaller risk estimates. In contrast to the 1980 NAS analysis, where the proportion of risk resulting from the dose squared term (e.g., C_3 in equation c of Table 8.2-1) was constrained to positive values, the linear quadratic function (which agrees best with Nagasaki cancer incidence data) has a negative coefficient for the dose squared term (Wa83). Although this negative coefficient is small and indeed may not be significant, the computational result is a larger linear term that leads to higher risk estimates at low doses than would be estimated using a simple linear model (Wa83). Preliminarily, the BEIR-3 analyses of mortality, which were not restricted to positive coefficients of the dose squared terms, yielded similar results.

Differences in the estimated cancer risk introduced by the choice of the risk projection model are also appreciable. As pointed out above, the 1980 NAS analysis indicates that relative risk estimates exceed absolute risk estimates by about a factor of 3, Table 8.2-1. However, relative risk estimates are quite sensitive to how the risk resulting from exposure during childhood persists throughout life. This question is addressed in Section 8.2.6 below, where we compare risk estimates made by the 1972 and 1980 NAS BEIR Committees with those of the ICRP and UNSCEAR.

8.2.6 Comparison of Cancer Risk Estimates for Low-LET Radiation

A number of estimates of the risk of fatal cancer following lifetime exposure are compared in Table 8.2-2. Although all of these

Table 8.2-2. A comparison of estimates of the risk of fatal cancer from a lifetime exposure at 1 rad/year (low-LET radiation)

	Cases per 10^6 person rad	Projection Model
BEIR-1 (NAS72)(a)	667	Relative Risk
BEIR-1 (NAS80)(b)	568	Relative Risk
BEIR-3 (NAS80)(b)(c)	403	Relative Risk
BEIR-3 (NAS80)(d)	169	Relative Risk
BEIR-3 (NAS80)(b)	158	Absolute Risk
BEIR-1 (NAS80)(b)	115	Absolute Risk
BEIR-3 (NAS80)(d)	67	Absolute Risk
UNSCEAR (UNSCEAR77)(e)	200-300	None--high dose > 100 rad
UNSCEAR (UNSCEAR77)(e)	75-175	None--low dose/dose rate
ICRP (ICRP77)	125	None--occupational - low dose/dose rate
CLM (Ch83)	100-440	UNSCEAR77 without A-bomb data

(a) BEIR-1 relative risk model.
(b) Table V-4 in NAS80, linear dose response.
(c) L-L absolute risk model for bone cancer and leukemia; $\overline{\text{L-L}}$ relative risk model for all other cancer.
(d) Table V-4 in NAS80, linear-quadratic dose response.
(e) Paragraphs 317 and 318 in UNSCEAR77.

risk estimates assume a linear response function, they differ considerably because of other assumptions. In contrast with absolute risk estimates, which have increased since the first NAS report (BEIR-1) was prepared in 1972 (NAS72), the 1980 NAS BEIR-3 Committee's estimates of the relative risk, as shown in Table 8.2-2, have decreased relative to those in the BEIR-1 report. This illustrates the sensitivity of risk projections to changes in modeling assumptions. In the NAS80 report, the relative risk of solid cancer observed for ages 10 to 19 was substituted for the considerably higher relative risk observed for those exposed during childhood, ages 0 to 9. In addition, the relative risk coefficients used in the BEIR-3 analysis are based on excess cancer in the Japanese A-bomb survivors compared to U.S. population cancer mortality rates. In the 1972 NAS report this excess was compared to cancer mortality in Japan. Moreover, the difference introduced by these two changes, particularly the former, is somewhat greater than indicated in the 1980 NAS report. The relative risk estimate attributed to the

BEIR-1 Committee in the NAS 1980 report is incorrect. Therefore, two BEIR-1 relative risk estimates are listed in Table 8.2-2: the risk estimate in NAS80 attributed to the BEIR-1 Committee and an estimate based on the risk coefficients in NAS72. The NAS 1980 estimate did not use the relative risk coefficient for childhood exposure given in the BEIR-1 report, which for solid cancers is a factor of 10 larger than adult values (p. 171 in NAS72), but rather used the adult risk for all ages including children. The estimate in Table 8.2-2 labeled NAS72 uses the relative risk coefficients actually given in the BEIR-1 report.

By comparing the three relative risk estimates in Table 8.2-2, it is apparent that the relative risk estimates are fairly sensitive to the assumptions made as to what extent the observed high relative risk of cancer from childhood exposure continues throughout adult life. The Life Span Study (Kab82) indicates that the high-risk adult cancer caused by childhood exposures is continuing, although, perhaps, not to the extent predicted by the NAS BEIR-1 Committee in 1972.

The major reason that the risk estimates in Table 8.2-2 differ is because of the underlying assumption in each set of risk estimates. The NAS BEIR estimates are for lifetime exposure and lifetime expression of induced cancers (NAS72, NAS80). Neither the age distribution of the population at risk nor the projection models (if any) have been specified by either the UNSCEAR (UNSCEAR77) or the ICRP (ICRP77). UNSCEAR apparently presumes the same age distributions as occurred in the epidemiological studies they cited, mainly the A-bomb survivors, and a 40-year period of cancer expression. The ICRP risk estimates are for adult workers, presumably exposed between ages 18 and 65, and a similar expression period. These are essentially age-independent absolute risk models with less than lifetime expression of induced cancer mortality. For these reasons alone, risks estimated by ICRP and UNSCEAR are expected to be smaller then those made on the basis of the BEIR-3 report.

The last entry in Table 8.2-2 (Chb83) is of interest because it specifically excludes the A-bomb survivor data based on T65 dose estimates. The authors reanalyzed the information on radiogenic cancer in UNSCEAR77 so as to exclude all data based on the Japanese experience. Their estimate of fatalities ranges from 100 to 440 per 10^6 person rad for high doses and dose rates. As indicated in Table 8.2-2, this is somewhat greater than but comparable to the UNSCEAR estimate, which includes the A-bomb survivor data. The mean number of fatalities given in Chb83 is 270 per 10^6 person rem, which is nearly identical to the value EPA has used for a linear dose response model--280 fatalities per 10^6 person rad (see below).

8.2.7 EPA Assumptions about Cancer Risks Resulting from Low-LET Radiations

EPA estimates of radiation risks are based on presumed linear and linear quadratic dose response functions. We believe these are the most credible dose response functions for estimating risks to exposed populations. Use of the BEIR-3 linear quadratic model is equivalent, at low dose, to using a dose rate effectiveness factor of 2.5.

Except for leukemia and bone cancer, where we use a 25-year expression period for radiogenic cancer, we use a lifetime expression period, as was done in the NAS report (NAS80). Because the most recent Life Span Study Report (Kab82) indicates that absolute risks for solid cancers are continuing to increase 33 years after exposure, the 1980 NAS Committee choice of a lifetime expression period appears to be well founded. We do not believe that limiting cancer expression to 40 years (as has been done by the ICRP and UNSCEAR) is compatible with the continuing increase in solid cancers that has occurred among irradiated populations (Kab82). Analyses of the spondylitic data have led others to similar conclusions (Sm78).

To project the number of fatalities resulting from leukemia and bone cancer, EPA uses an absolute risk model, a minimum induction period of 2 years, and a 25-year expression period. To estimate the number of fatalities resulting from other cancers, EPA uses the arithmetic average of absolute and relative risk projection models. For these cancers, we assume a 10-year minimum induction period and expression of radiation-induced cancer for the balance of an exposed person's lifetime after the minimum induction period.

8.2.8 Methodology for Assessing the Risk of Radiogenic Cancer

EPA uses a life table analysis to estimate the number of fatal radiogenic cancers in an exposed population of 100,000 persons. This analysis considers not only death resulting from radiogenic cancer but also the probabilities of other competing causes of death which are, of course, much larger and vary considerably with age (Bu81, Co78). Basically, it calculates for ages 0 to 110 the risk of death resulting from all causes by applying the 1970 mortality data from the National Center for Health Statistics (NCHS75) to a cohort of 100,000 persons. Additional information in the details of the life table analysis are provided in Addendum B. It should be noted that a life table analysis is required to use the age-dependent risk coefficients in the BEIR-3 report. For relative risk estimates, we use age-specific cancer mortality data also provided by NCHS (NCHS73). The EPA computer program we use for the life table analysis was furnished to the NAS BEIR-3 Committee by EPA and was used by the Committee to prepare their risk estimates. Therefore, we are sure that the population base and calculations are the same in both the NAS and EPA analyses.

To project the observed risks of most solid radiogenic cancers beyond the period of current observation, we use both absolute and relative risk models, but usually present an arithmetic average based on these projections. Use of a single estimate instead of a range of values does not mean that our estimate is precise. As indicated in Table 8.2-2, the range of estimated fatal cancers resulting from the choice of a particular projection model and its internal assumptions is about a factor of three. Although we think it is likely that the relative risk model is the best projection model for most solid cancers, it has been tested rigorously only for lung and breast cancer (Lab78). Until it has more empirical support, we prefer to use an average risk based on both projection models. A second reason for this choice is to avoid overly conservative risk estimates caused by the compounding of multiplicative conservative assumptions.

To estimate the cancer risk from low-LET, whole-body, lifetime exposure with the linear model, we use the arithmetic average of relative and absolute risk projections (the BEIR-3 $\overline{\text{L-L}}$ model) for solid cancers and an absolute risk projection for leukemia and bone cancer (the BEIR-3 L-L model). For dose to the whole body, this yields an estimated 280 fatalities per million person rad. For the BEIR-3 linear quadratic model, which is equivalent to assuming a DREF of 2.5, a low-LET whole-body dose yields an estimated life risk of about 110 fatalities per million person rad.

These risk estimates are not unduly conservative. More than 235 of the 280 fatalities estimated with the BEIR-3 linear model result from cancers in soft tissues for which we have used the BEIR-3 $\overline{\text{L-L}}$ model. As explained on page 187 of that report (NAS80), the $\overline{\text{L-L}}$ model is not derived from the observed risk of solid cancers alone but rather includes parameters based on the Committee's analysis of the leukemia mortality data. Therefore, as outlined in 8.5, the BEIR-3 Committee's analysis of the Japanese leukemia data depended heavily on the assumption that most of the leukemia observed at Hiroshima was caused by neutrons. In contrast, Table V-30 in the BEIR-3 report (NAS80) estimates the risk of cancer *incidence* in soft tissues directly, without the additional assumptions contained in the BEIR-3 $\overline{\text{L-L}}$ model. By using the weighted incidence mortality ratios given in Table V-15 (NAS80), the results given in Table V-30 (NAS80) can be expressed in terms of mortality, to yield (for lifetime exposure) an absolute risk estimate of about 200 fatalities per 10^6 person rad and about 770 fatalities per 10^6 person rad when a relative risk projection model is used to estimate lifetime risk. The arithmetic mean of the fatalities projected by these two models is almost 500 per 10^6 person rad, more than twice as many fatal soft tissue cancers as predicted by the BEIR-3 $\overline{\text{L-L}}$ model and about five times as many as estimated using the BEIR-3 linear quadratic model.

By a whole-body dose, we mean a uniform dose to every organ in the body. In practice, such exposure situations seldom occur, particularly for ingested or inhaled radioactivity. The next section describes how we apportion this risk estimate for whole-body exposure when considering the risks following the exposure of specific organs.

8.2.9 Organ Risks

For most sources of environmental contamination, inhalation and ingestion of radioactivity are more common than direct exposure. In many cases, depending on the chemical and physical characteristics of the radioactive material, inhalation and ingestion result in a non-uniform distribution of radioactive materials within the body so that some organ systems receive much higher doses than others. For example, iodine isotopes concentrate in the thyroid gland, and the dose to this organ can be orders of magnitude larger than the average dose to the whole body.

Fatal Cancer at Specific Sites

To determine the probability that fatal cancer occurs at a particular site, we have performed life table analyses for each cancer type using the information on cancer incidence and mortality in NAS80. For cancer other than leukemia and bone cancer we used NAS80 Table V-14 (Age Weighted Cancer Incidence by Site Excluding Leukemia and Bone Cancer) and NAS80 Table V-15, which lists the BEIR Committee's estimates of the ratio of cancer fatality to cancer incidence for these various organs. The proportions of leukemia and fatal bone cancer caused by low-LET radiation were estimated using the results given in Tables V-17 and V-20 of NAS80. Normalized results, which give the proportion of fatal cancer caused by radiogenic cancer at a particular site, are listed in Table 8.2-3. As noted above, these proportions are assumed to be the same for the BEIR-3 linear quadratic dose respose model.

Information on the proportion of fatal cancers resulting from cancer at a particular organ is not precise. One reason is that the data in NAS80 (and in Table 8.2-3) are based on whole-body exposures, and it is possible that the incidence of radiogenic cancer varies depending on the number of organs exposed. Except for breast and thyroid cancer, very little information is available on radiogenic cancer resulting from exposure of only one region in the body. Another reason is that most epidemiology studies use mortality data from death certificates, which often provide questionable information on the site of the primary cancer. Moreover, when the existing data are subdivided into specific cancer sites, the number of cases becomes small, and sampling variability is increased. The net result of these factors is that numerical estimates of the total cancer risk are more reliable than those for most single sites.

The 1977 UNSCEAR Committee's estimated risks (UNSCEAR77) to different organs are shown in Table 8.2-4. For all of the organs except the breast, a high and low estimate was made. This range varies by a factor of two or more for most organs, Table 8.2-4. Other site-specific estimates show a similar degree of uncertainty (Kab82), and it is clear that any system for allocating the risk of fatal cancer on an organ-specific basis is inexact. Table 8.2-5 compares proportional risks by the NAS BEIR-3 Committee, UNSCEAR, and the ICRP. ICRP Report 26 provides organ-specific weights for assessing combined genetic and cancer risks, due to occupational exposure (ICRP77). In Table 8.2-5, we have renormalized ICRP risks so that they pertain to cancer alone.

Considering that the cancer risk for a particular site is usually uncertain by a factor of two or more, as indicated by the range of UNSCEAR estimates in Table 8.2-4, we would not expect perfect agreement

Table 8.2-3. Proportion of the total risk of fatal radiogenic cancer resulting from cancer at a particular site

Site	Proportion of Total Risk(a)
Lung	0.21
Breast(b)	0.13
Red bone marrow(c)	0.16
Thyroid	0.099
Bone	0.009
Liver	0.085
Stomach	0.084
Intestines	0.039
Pancreas	0.058
Kidneys and urinary tract	0.025
Other(d)	0.11

(a) NAS80 - Lifetime exposure and cancer expression; results are rounded to two figures.

(b) Average for both sexes.

(c) Leukemia.

(d) Total risk for all other organs, including the esophagus, lymphatic system, pharynx, larynx, salivary gland, and brain.

Table 8.2-4. UNSCEAR estimates of cancer risks at specified sites

Site	Fatalities (10^6/person rad)	Average (10^6/organ rad)	Proportion of Total Risk
Lung	25-50	37.5	0.24
Breast[a]	25	25.0	0.16
Red bone marrow[b]	15-25	20.0	0.13
Thyroid	5-15	10.0	0.065
Bone	2-5	3.5	0.023
Liver	10-15	12.5	0.081
Stomach	10-15	12.5	0.081
Intestines	14-23	18.5	0.12
Pancreas	2-5	3.5	0.023
Kidneys and urinary tract	2-5	3.5	0.023
Other[c]	4-10	7.0	0.046

[a] Average for both sexes.
[b] Leukemia.
[c] Includes esophagus and lymphatic tissues.
Source: (UNSCEAR77).

Table 8.2-5. Comparison of proportion of the total risk of radiogenic cancer fatalities by body organ

Site	NAS80[a]	UNSCEAR (UNSCEAR77)	ICRP77[b]
Lung	.21	.24	.16
Breast	.13	.16	.20
Red bone marrow	.16	.13	.16
Thyroid	.099	.065	.04
Bone	.009	.023	.04
Liver	.085	.081	(.08)[c]
Stomach	.084	.081	(.08)
Intestines	.039	.12	(.08)
Pancreas	.058	.023	(.08)
Kidneys and urinary tract	.025	.023	(.08)
Other	.11[d]	.046	--

[a] Lifetime exposure and cancer expression.
[b] Normalized for risk of fatal cancer (see text).
[c] Five additional organs that have the highest dose are assigned 0.08 for a total of 0.4.
[d] Others include esophagus, lymphatic system, pharynx, larynx, salivary gland, and brain.

in apportionment of total body risks. Table 8.2-5, however, does indicate reasonable agreement among the three sets of estimates considered here.

The differences between the proportions of the total risk of fatal cancer shown in Table 8.2-5 are, for the most part, small in comparison to their uncertainty. We have used the BEIR-3 organ risks in preference to those made by other groups such as UNSCEAR or the ICRP for several reasons. BEIR estimates of organ risk are based on a projection of lifetime risk using age-specific risk coefficients, rather than just observations to date. Moreover, the 1980 BEIR Committee considered cancer incidence data as well as mortality data. This gives added confidence that the diagnostic basis for their estimates is correct. And, finally, because we apply these proportional organ risk estimates to the NAS80 cancer risk estimates for whole-body exposures, we believe it is consistent to use a single set of related risk estimates. The way we have used NAS80 to estimate mortality resulting from cancer at a particular site is outlined in the next section.

8.2.10 Methodology for Calculating the Proportion of Mortality Resulting from Leukemia

Application of NAS80 to particular problems is straightforward but requires some familiarity with the details of that report. In this section we provide sample calculations based on the BEIR-3 linear dose response model for the case of fatal leukemia resulting from irradiation of the bone marrow throughout an average person's lifetime. We then compared this number to the average number of all fatal radiogenic cancers to obtain the proportion caused by leukemia, as shown in Table 8.2-3.

The NAS80 estimates in Table 8.2-3 differ from the others in that they include both a consideration of age at exposure and a full expression of radiogenic cancer resulting from lifetime exposure. For example, Table V-17 (NAS80) gives explicit age- and sex-dependent mortality coefficients for leukemia and bone cancer together.

The ratio of leukemia to bone cancer fatalities is given by the coefficient in the dose response relationship listed in Table V-17, i.e. 2.24/0.05. For lifetime exposure at a dose rate of one rad per year, Table V-17 lists 3,568 leukemia (and bone) deaths per 10^6 males and 2,709 deaths per 10^6 females (NAS80). Using a male-female birth ratio of 1.05 to 1.0, this averages to 3,149 fatal cancers per million persons in the general population. The total person rad causing these excess fatalities is the product of one rad per yr, 10^6 persons, and 70.7 years (the average age of this population at death). Dividing the total number of fatalities by this product yields 44.5 fatalities per 10^6 person rad of which about 43.5 are caused by leukemia. As noted above, for total body exposure, the average of the absolute and relative risk projection models yielded 280 per 10^6 person rad. Therefore, P, the proportion of the whole-body risk caused by the lifetime risk of a leukemia death resulting from lifetime exposure of the red bone marrow is:

$$P = \frac{43.5}{280} = 0.16 \quad \text{(cf. with Table 8.2-3)} \qquad (8\text{-}1)$$

To obtain the proportional mortality for other cancers, we have used the site-specific, age-dependent risk coefficients in Table V-14 (NAS80) and the mortality ratios in Table V-15 to calculate the risk of fatal cancer from lifetime exposure at one rad per year (for each sex) and proceeded as in the example for leukemia outlined above.

To apply the data shown in Table 8.2-3 to a particular organ we multiply the average of the relative and absolute lifetime risk estimates for whole-body lifetime exposure for a linear dose response, 280 fatalities per 10^6 person rad, and 112 fatalities per 10^6 person rad for a linear quadratic response by the proportional mortality for that cancer. For example, using the linear model, a one rad dose (low-LET) to the kidney (urinary tract) resulting from lifetime exposure is estimated to cause a lifetime probability of death caused by radiogenic cancer that is equal to (.025) x (280×10^6) or 7×10^{-6}, i.e., 7 chances in a million.

Iodine-131 has been reported to be only 1/10 as effective as X-rays or gamma rays in inducing thyroid cancer (NAS72, NCRP77). For this cancer a linear dose response and a DREF of 10 is used in calculating lifetime probability of death. For example, the risk from a one rad dose to the thyroid from exposure to iodine-131 or iodine-129 is calculated as follows: (0.099) x (0.10) x (280×10^{-6}) or 2.8×10^{-6}, about 3 chances in a million.

8.2.11 Cancer Risks Due to Age-Dependent Doses

As noted in Chapter 7, almost all of the dose models we have used are based on ICRP "Reference Man". (An exception is the case of radon progeny where we use an age-dependent "exposure" model, see below.) ICRP dosimetric models are appropriate for adult workers and do not take into account differences resulting from the changes in physiological parameters between children and adults, e.g., intake rates, metabolism, and organ size. Although it is difficult to generalize for all radionuclides, in some cases these differences tend to counterbalance each other. For example, the ratio of minute volume to lung mass is relatively constant with age, so that the ICRP adult model for inhaled insoluble materials provides a reasonably good estimate of the average annual dose throughout life.

An exception is the thyroid where the very young have a relatively high uptake of radioiodine into a gland which is much smaller than the adult thyroid, as noted in Table 7.5-1. This results in a larger childhood dose and an increased risk which persists throughout life. Since this is a worse case situation, we have examined it with some care, using the age-specific risk coefficients for thyroid cancer in Table V-14 of the BEIR-3 report (NAS80) and the age-dependent dose model in ORNL84. For iodine-131 ingestion, the estimated lifetime time risk is increased by a factor of 1.56 due to the 30 percent increase in lifetime dose over that obtained with the ORNL adult model, cf. Chapter 7. Results are about the same for inhalation of iodine-131, the estimated lifetime risk of fatal thyroid cancer by a factor of 1.63 for ORNL's age-dependent dose estimate.

As noted in Chapter 7, use of an age-dependent dosimetry for other radionuclides has yielded much smaller increased doses relative to adult models and therefore has little effect on estimates of lifetime risk. In particular, the lung dose and risk resulting from the inhalation of insoluble alpha particle emitters is nearly unchanged. The lifetime dose for an age-dependent dose model is only 1.09 times greater than that calculated using an adult model (Chapter 7); the lifetime risk of lung cancer for this age-dependent model is a factor of 1.16 greater than we calculate for life exposure with the adult-only model. This is important because, as noted in Volume II of this BID, such radionuclides are the major cause of increased cancer resulting from the emission of radionuclides into air.

EPA's age-dependent exposure model for radon progeny outlined in Section 8.2 yields a 12 percent greater exposure than a lifetime exposure using just the adult intake. The lifetime risk of lung cancer for the more realistic exposure pattern is 22 percent greater. We have concluded that with the possible exception of some iodine isotopes, e.g., iodine-131, the use of the ICRP dosimetry does not contribute a significant source of uncertainty in this rulemaking. We recognize, however, that good physiological data for children is not available for many radionuclides and that there may be other exceptions. These exceptions will not include inhaled insoluble alpha-emitting particulates.

8.3 Fatal Cancer Risk Resulting from High-LET Radiations

In this section we explain how EPA estimates the risk of fatal cancer resulting from exposure to high-LET radiations. In some cases, ingestion and inhalation of alpha particle emitting radionuclides can result in a relatively uniform exposure of the body organs by high-LET radiations. Unlike exposures to X-rays and gamma rays where the resultant charged particle flux results in linear energy transfers (LET) of the order of 0.2 to 2 keV per micron in tissue, 5 MeV alpha particles result in energy deposition at a track average rate of more than 100 keV per micron. High-LET radiations have a larger biological effect per unit dose (rad) than low-LET radiations. How much greater depends on the particular biological endpoint being considered. For cell killing and other readily observed endpoints, the relative biological effectiveness (RBE) of high-LET alpha radiations is often ten or more times greater than low-LET radiations.

8.3.1 Quality Factors for Alpha Particles

Charged particles have been assigned quality factors, Q, to account for their efficiency in producing biological damage. Unlike an RBE value, which is for a specific and well-defined endpoint, a quality factor is based on an average overall assessment by radiation protection experts of potential harm of a given radiation relative to x or gamma radiation. In 1977, the ICRP assigned a quality factor of 20 to alpha particle irradiation from radionuclides (ICRP77). The reasonableness of this numerical factor for fatal radiogenic cancers at a particular site is not well known, but it is probably conservative for all sites and highly conservative for some.

The dose equivalent, in the unit rem, is the dose, in rad, times the appropriate quality factor for a specified kind of radiation. For the case of internally deposited alpha particle emitters the dose equivalent from a one-rad dose is equal to 20 rem. It should be noted that prior to ICRP Report 26 (ICRP77) the quality factor for alpha particle irradiation was ten. That is, the biological effect from a given dose of alpha particle was estimated to be ten times that from an acute dose of low-LET X-rays or gamma rays of the same magnitude in rad. The ICRP decision to increase this quality factor to 20 followed from their decision to estimate the risk of low-LET radiations, in occupational situations, on the assumption that biological effects were reduced at low dose rates for low-LET radiation. There is general agreement that dose rate effects do not occur for high-LET (alpha) radiations. The new ICRP quality factor for alpha particles of 20 largely compensates for the fact that their low-LET risks are now based on an assumed dose rate reduction factor of 2.5. This DREF has been used in preparing EPA estimates of the risk per rad for alpha particle doses described below, in 8.3.3.

In 1980 the ICRP published a task group report "Biological Effects of Inhaled Radionuclides" which compared the results of animal experiments on radiocarcinogenesis following the inhalation of alpha particle and beta particle emitters (ICRP80). The task group concluded that "the experimental animal data tend to support the decision by the ICRP to change the recommended quality factor from 10 to 20 for alpha radiation."

8.3.2 Dose Response Function

In the case of high-LET radiation, a linear dose response is commonly observed in both human and animal studies and that the response is not reduced to low dose rates (NCRP80). Some data on human lung cancer indicate that the carcinogenic response per unit dose of alpha radiation is higher at low doses than higher ones (Ara81, Hob81, Wh83); in addition, some studies with animals show the same response (Cha81, U182). We agree with the NAS BEIR-3 Committee that, "For high-LET radiation, such as from internally deposited alpha-emitting radionuclides, the linear hypothesis is less likely to lead to overestimates of the risk and may, in fact, lead to underestimates" (NAS80). However, at low doses, departures from linearity are small compared to the uncertainty in the human epidemiological data, and we believe a linear response provides an adequate model for evaluating risks in the general environment.

A possible exception to a linear response is provided by the data for bone sarcoma (but not sinus carcinoma) among U.S. dial painters who have ingested alpha-emitting radium-226 (NAS80). These data are consistent with a dose squared response (Ro78). Consequently, the NAS BEIR-3 Committee estimated bone cancer risk on the basis of both linear and quadratic dose response functions. However, as pointed out in NAS80, the number of U.S. dial painters at risk who received less than 1000 rad was so small that the absence of excess bone cancer at low doses is not statistically significant. Therefore, the consistency of these data with a quadratic (or threshold) response is not remarkable and, perhaps, not relevant to evaluating risks at low doses. In contrast to the dial painter data, the incidence of bone cancer following radium-224 irradiation, observed in spondylitics by Mays and Spiess (Mab83, NAS80), in a larger sample at much lower doses, is consistent with a linear response. Therefore, for high-LET radiations the EPA has used a linear response function to evaluate the risk of bone cancer.

Closely related to the choice of a dose response function is what effect the rate at which a dose of high-LET radiation is delivered has on its carcinogenic potential. This is a very active area of current research. There is good empirical evidence, from both human and animal studies, that repeated exposures to radium-224 alpha particles is five times more effective in inducing bone sarcomas than a single exposure which delivers the same dose (Mab83, NAS80). The 1980 NAS BEIR Committee took this into account in their estimates of bone cancer fatalities, which EPA is using. We do not know to what extent, if any, a similar enhancement of carcinogenicity may occur for other cancers resulting from internally deposited alpha particle emitters. Nevertheless, we believe that the ICRP quality factor of 20 is conservative, even at low dose rates.

8.3.3 Assumptions Made by EPA for Evaluating the Dose from Alpha Particle Emitters

We have evaluated the risk to specific body organs by applying the ICRP quality factor of 20 for alpha radiations to the risk estimates for low dose rate low-LET radiations described in Section 8.2.9. For some organs this quality factor may be too conservative. Several authors have noted that estimates of leukemia based on a quality factor of 20 for bone marrow irradiation overpredicts the observed incidence of leukemia in persons receiving Thorotrast (thorium oxides) (Moa79) and in the U.S. radium dial painters (Spb83). Nevertheless, in view of the paucity of applicable human data and the uncertainties discussed above, the ICRP quality factor provides a reasonable and prudent way of evaluating the risk resulting from alpha emitters deposited within body organs.

All of the EPA risk estimates for high-LET radiations are based on a linear dose response function. For bone cancer and leukemia we use the absolute risk projection model described in the previous section. For other cancers we use the arithmetic average of relative and absolute risk projections.

Table 8.3-1 indicates EPA's estimates of the risk of fatal cancer resulting from a uniform organ dose in various organs from internally deposited alpha particles. It was prepared by multiplying the average risk (based on the linear model for a uniformly distributed whole-body dose of low-LET radiation and, unlike the DBID (EPA83a), a dose rate effectiveness factor of 2.5) by a quality factor of 20 and then apportioning this risk by organ, as indicated in Table 8.3-1. These estimates are for lifetime doses at a constant dose rate. This procedure

Table 8.3-1. Estimated number of cancer fatalities from a lifetime exposure to internally deposited alpha particle emitters

Site	Proportional Risk[a]	Fatalities per 10^6 organ rad[b]
Lung	.21	460
Breast[c]	.13	290
Red bone marrow[d]	.16	350
Thyroid	.099	220
Bone[e]	.009	20
Liver	.085	190
Stomach	.084	190
Intestine	.039	90
Pancreas	.058	130
Kidneys and urinary tract	.025	55
Other-Sum (total)	.11	250

[a] Proportion of whole body risk from Table 8.2-3.

[b] Rounded to two figures. Note that these estimates are 2.5 times smaller than those used in preparing the DBID.

[c] Average for both sexes.

[d] Leukemia.

[e] Bone endosteum as defined in ICRP-30 (ICRP79).

was not followed for bone cancer. As outlined above, the risk estimate for this cancer in the BEIR-3 report (NAS80) is based on data for high-LET (alpha) radiation.

Some readers may note that the risk estimate in Table 8.3-1, about 20 bone cancer fatalities per 10^6 person rad, is less than the 27 fatalities listed in Table A-27 of NAS80 for alpha particles. This is because the analysis in Appendix A of NAS80, but not Chapter V of that report, assumes that in addition to a 2-year minimum induction period, 27 years are available for cancer expression. This is usually not the case for doses received beyond middle age. Hence, the estimated lifetime risk is smaller when it is based on a life table analysis that considers lifetime exposure in conjunction with death from all causes.

In the next section, we describe how we estimate the risk resulting from inhalation of alpha-emitting radon progeny, a situation where the organ dose is highly nonuniform.

8.4 Estimating the Risk Resulting from Lifetime Population Exposures from Radon-222 Progeny

EPA estimates of the risk of lung cancer resulting from inhaled radon progeny do not utilize the dosimetric approach, outlined above, but are based on what is sometimes called an epidemiological approach. In this approach the amount of excess human lung cancer in groups known to have been exposed to radon progeny is determined.

When radon-222 (a radioactive atomic gas) decays, a number of short half-life radionuclides, principally polonium-218, lead-214, bismuth-214, and polonium-214, are formed that attach to inhalable dust particles in air. When inhaled, the dust containing the radon progeny plates out on the surfaces of the larger bronchi of the lung. Since two of these radionuclides decay by alpha particle emission, bronchial epithelium is irradiated by high-LET radiation. A wealth of data indicate that a range of exposures to the bronchial epithelium of underground miners causes an increase in bronchial lung cancer, both in smoking and in nonsmoking miners. Two recent reviews on the underground miner experience are of particular interest. The 1980 NAS BEIR-3 Report (NAS80) contains a review of the epidemiological studies on these miners. Thomas and McNeil (Th82) reanalyze many of these epidemiological studies in a consistent fashion so that the modeling assumptions are the same for all of the data sets.

Although considerable progress has been made in modeling the deposition of particulate material in the lung (Hac82, Jaa80, Jac81), it is not yet possible to adequately characterize the bronchial dose delivered by alpha radiation from radon-222 progeny attached to dust particles. This is because of the lack of knowledge concerning the kinds of cells in which bronchial cancer is initiated (Mc78) and the depth of these cells in the bronchial epithelium. Current estimates of the dose actually causing radiogenic cancer resulting from inhaled radon-222 progeny are based on average doses that may or may not be relevant. Until more reliable estimates of the bronchial dose become available, we are following the precedents set in the 1972 and 1980 NAS reports (NAS72, NAS80) and are estimating the risk resulting from radon-222 progeny on the basis of exposure rather than dose *per se*. This is called the epidemiological approach, i.e., risk is estimated on the basis of observed cancers following occupational exposure to radon progeny.

8.4.1 Characterizing Exposures to the General Population vis-a-vis Underground Miners

Exposures to radon under working conditions are commonly reported in a special unit called the working level (WL). One working level is any concentration in air of short half-life radon-222 progeny having 1.3 x 10^5 MeV per liter of potential alpha energy (FRC67). This unit was developed because the concentration of specific radon progeny depends on ventilation rates and other factors. A working level month (WLM) is the unit used to characterize a miner's exposure to one working level of radon progeny for a working month of about 170 hours. Because the results of epidemiological studies are expressed in units of WL and WLM, we outline below how they can be interpreted for members of the general population exposed to radon progeny.

For a given concentration of radon progeny, the amount of potential alpha energy inhaled in a month by a member of the general population is more than that received in a miner's working month. These individuals are exposed longer, up to 24 hours per day, 7 days a week. However, the average amount of air inhaled per minute (minute volume) by a member of the general population is less than the amount for a working miner when such activities as sleeping and resting are taken into account. To compare the radon progeny exposure of a working miner to a member of the general population, we have calculated the amount of potential alpha energy each inhales per year.

We have assumed that (averaged over a work day) a miner inhales 30 liters per minute. This average corresponds to about 4 hours of light activity and 4 hours of moderately heavy work per day (ICRP75). We recognize that the new ICRP radon model assumes a 20 liter per minute volume for miners, which corresponds to 8 hours of light activity per day (ICRP81). Although this may be appropriate for nuclear workers, studies of the metabolic rate of working miners clearly show that they are not engaged only in light activity (Spa56, ICRP75, NASA73). Therefore, we have chosen 30 liters as a more realistic estimate of their average minute volume. A working miner with this minute volume inhales 3.6 x 10^3 cubic meters in a working year of 2000 hours (ICRP79). One working level of radon-222 progeny is 2.08 x 10^{-5} Joules per cubic meter. Therefore, in a working year the potential alpha energy inhaled by a miner exposed to one working level is 7.5 x 10^{-2} Joules.

For adult males and females in the general population we follow the ICRP Task Group on Reference Man (ICRP75) in assuming an inhaled air volume of 2.3 x 10^4 liters per day for males and 2.1 x 10^4 liters per day for adult females, an average of 2.2×10^4 liters per day. This average volume results in 1.67×10^{-1} Joules per year of inhaled potential alpha energy from an exposure to one working level of radon-222 progeny for 365.25 days. Although it may be technically inappropriate to quantify the amount of potential alpha particle energy inhaled by a member of the general population in working level months, this amounts to an annual exposure equivalent to 27 WLM (26.7) to an adult member of the general population exposed 24 hours per day. For indoor exposure, we have assumed an occupancy factor of 0.75 so that an exposure to one WL results in an annual exposure equivalent to 20 WLM (EPA78) in terms of the amount of potential alpha energy actually inhaled.

Children have a smaller bronchial area than adults, which more than offsets their lower minute volume, so that the dose to their bronchi, for a given concentration of radon progeny, is greater. This problem has been addressed by Hofmann and Steinhausler (Hoa77). Their results indicate that exposures received during childhood are about 50 percent greater than adult exposures. We have used the information in (Hoa77) to prepare Table 8.4-1, which lists the age-dependent potential alpha energy exposure we have used in the risk assessments listed below.*

Table 8.4-1. Potential alpha energy inhaled during one year of exposure to one working level (2.08×10^{-5} Joules per cubic meter) as a function of age by a member of the general population(a)

Age (years)	Joules	WLM(a)
0-2	0.22	35
3-5	0.27	43
6-11	0.30	49
12-15	0.27	43
16-19	0.24	38
20-22	0.20	32
23 or more	0.17	27

(a)Assuming a WLM corresponds to 6.2×10^{-3} Joules of potential alpha particle energy inhaled (see text).
Source: (Hoa77).

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*The assumptions on minute volume, etc. for miners and the general population described above are the same as those used in the preparation of the EPA reports (EPA79,82,83a,b).

The results in Table 8.4-1 have been rounded to two significant figures. The larger exposure to children relative to adults increases the estimated mortality due to lifetime exposure from birth by about 20 percent.

We have also examined the exposure model described above in terms of the average dose delivered to bronchial tissue using the most detailed dose model available, the five-lobe lung model developed by Harley and Pasternack (Hac82). For the breathing patterns we have assumed for each group, the bronchial dose per WLM for working miners is 0.64 rad, and is 0.51 rad for an adult member of the general population (Had83). Therefore, we have concluded that the factors not included in our simple model, such as the fraction of unattached radon progeny, are not very important compared to other sources of uncertainty in our risk estimates.

8.4.2 The EPA Model

Since 1978, EPA has based risk estimates of cancer resulting from inhaled radon-222 progeny on a linear dose response function, a relative risk projection model, and a minimum induction period of 10 years. Lifetime risks are projected on the assumption that exposure to 1 WLM increases the age-specific risk of lung cancer by 3 percent over the age-specific rate in the U.S. population as a whole. The life table analysis described in the annex to this chapter is used to project this risk over a full life span.

The EPA model has been described in detail (EPA79, Elb79). In reviewing this model in terms of the more recent information described below, we have found that our major assumptions, linear response and relative risk projection, have been affirmed. The A-bomb survivor data clearly indicate that the absolute risk of radiogenic lung cancer has continued to increase among these survivors while their relative risk has remained reasonably constant (Kab82). The UNSCEAR, ICRP, and 1980 NAS Committee have continued to use a linear dose response to estimate the risk of lung cancer resulting from inhaled radon progeny. Thomas and McNeill's analysis (Th82) indicates that the use of linearity is not unduly conservative and may, in fact, underestimate the risk at low doses. As noted above, the 1980 NAS BEIR Committee reached a similar conclusion.

A major limitation of the EPA model is the uncertainty in the relative risk coefficient we have used, 3 percent increase per WLM. This value is based on the excess mortality resulting from lung cancer among exposed miners of various ages, many of whom smoked. Therefore, it is an average value for a mixed population of smokers and nonsmokers. Furthermore, the fact that smoking was more prevalent among some of the groups of miners studied than it is among the U.S. general population today, this may lead to an overly conservative risk estimate as discussed below.

In a recent paper, Radford and Remard (Ra84) reported on the results of a long-term study of Swedish iron miners who were exposed to radon progeny. This study is unique in that most of the miners were exposed to less than 100 WLM, and the risks to smokers and nonsmokers was considered separately. The absolute risk of the two groups was similar, 20 fatalities per 10^6 person WLM year for smokers compared to 16 for nonsmokers. The total number of lung cancer fatalities for nonsmokers is small; so that the estimate of 16 is not too reliable.

While absolute risks were comparable for the smoking and nonsmoking miners, relative risks were not. Nonsmokers have a much lower baseline incidence of lung cancer mortality than smokers. This resulted in a relative risk coefficient for nonsmoking exposed miners relative to unexposed nonsmokers that was four times larger than the relative risk coefficient for exposed smokers. However, this larger relative risk does not fully compensate for the lower base line incidence of nonsmokers. Therefore, this study of Swedish iron miners indicates that a 3 percent per WLM relative risk coefficient may be too conservative when appied to the population as a whole. Further follow-up of this and other mining groups may provide more reliable data on the risk to nonsmokers and we expect to incorporate separate consideration of smokers and non-smokers into EPA analyses as more data becomes available.

Although occupational exposures to pollutants other than radon-222 progeny are probably not important factors in the observed lung cancer risk for underground miners (Elb79, Th82, Mua83, Ra84), the use of occupational risk data to estimate the risk of a general population is far from optimal, as it provides no information on the effect of radon progeny exposures to children and women. Although we have continued to assume that the risk per unit exposure during childhood is no more effective than that occurring to adults, this assumption may not be correct. The A-bomb survivor data indicate that, in general, the risk from childhood exposure to low-LET radiation is greater and continues throughout life (Kab82). There are no specific data for lung cancer yet (Kab82). Another limitation of the underground miner data is the absence of women in the studied populations. The A-bomb survivor data indicates that women are as sensitive as men to radiogenic lung cancer even though, on the whole, they smoke less (Pr83). These data are not conclusive, however.

8.4.3 Comparison of Risk Estimates

Several estimates of the risk resulting from radon progeny have been published since the EPA model was developed. One of particular interest was expounded by the BEIR Committee (NAS80). The BEIR-3 Committee formulated an age-dependent absolute risk model with increasing risk for older age groups. The Committee estimates of the risk per WLM for various ages are listed on page 325 in NAS80, and their estimated minimum induction period for lung cancer following exposure on page 327. We have used these data, summarized in Table 8.4-2, to calculate the lifetime risk of lung cancer mortality from lifetime exposure to persons in the general population by means of the same life table analysis used to calculate other EPA risk estimates.

Table 8.4-2. Age-dependent risk coefficients and minimum induction period for lung cancer resulting from inhaling radon-222 progeny

Age (yr)	Excess (cases per 10^6 WLM person years)	Minimum induction period (years)
0-14	0	25
15-34	0	25-15
35-49	10	10
50-64	20	10
65 or greater	50	10

Source: NAS80.

It should be noted that the zero risk shown in Table 8.4-2 for those under 35 years of age at exposure does not mean no harm occurs but rather that it is expressed after the person is more than 35 years old, i.e., only after the minimum induction period. The sequence of increasing risk with age shown in Table 8.4-2 is not unlike the increase in lung cancer with age observed in unexposed populations, so that the pattern of excess risk over time is similar to that found using a relative risk projection model.

Thomas and McNeil conducted a thorough analytical investigation of lung cancer among uranium miners for the AECB of Canada (Th82). These investigators tested a number of risk models on all of the epidemiological studies that contained enough data to define a dose response function. They concluded that, for males, a 2.3 percent increase in lung cancer per WLM and a relative risk projection model were more consistent with the excess lung cancer incidence observed in underground miner groups than other models they tested. This is the only analysis we are aware of that treated each data set in consistent fashion and utilized modern epidemiological techniques, such as controlling, to the extent possible, for age at exposure and duration of follow-up.

The AECB risk estimates for lifetime exposure to a general population along with EPA, NAS, UNSCEAR, ICRP, and NCRP estimates of the risk of lung cancer resulting from inhaled radon progeny are listed in Table 8.4-3. The AECB estimate for lifetime exposure to Canadian males is 830 fatalities per million person WLM (Th82). In Table 8.4-3 this estimate has been adjusted for the U.S. 1970 population of males and females.

Table 8.4-3. Risk estimate for exposures to radon progeny[a]

Organization	Fatalities per 10^6 person WLM	Exposure Period	Expression Period
EPA[b]	760	Lifetime	Lifetime
NAS BEIR-3[b]	730	Lifetime	Lifetime
AECB[c]	600	Lifetime	Lifetime
ICRP	150-450	Working Lifetime	30 years
UNSCEAR	200-450	Lifetime	40 years
NCRP[d]	130	Lifetime	Lifetime

(a)The number of fatalities per 10^6 person WLM listed for EPA and NAS80 in this table differs from figures we have previously published (e.g., EPA83b) because we have now included, correctly we believe, the increased potential alpha energy exposure during childhood in the denominator of this ratio. Our risk estimates for various sources of radon in the environment have not changed, because all were calculated via a life table analysis yielding deaths per 100,000 exposed, not deaths per person WLM.
(b)Assumes increased exposure during childhood, Table 8.4-1.
(c)Adjusted for U.S. general population, see text.
(d)Assumes that risk diminishes exponentially with a 20-year half-life.
Source: EPA, EPA83b; NAS BEIR-3, NAS80; AECB, Th82; ICRP, ICRP81; UNSCEAR, UNSCEAR77; NCRP, NCRP84, USRPC80.

The agreement between the EPA, BEIR-3, and the AECB estimates shown in Table 8.4-3 is not unexpected. Each estimate is based on lifetime exposure and lifetime expression of the incurred risk. In contrast, the three lower risk estimates in Table 8.4-3 do not explicitly include these factors.

The ICRP estimates are for occupational exposure to working adults. The larger ICRP estimate is based on their epidemiological approach, that is, the exposure to miners in WLM and the risk per WLM observed in epidemiological studies of underground miners. The ICRP epidemiological approach assumes an average expression period of 30 years for lung cancer. Children, who have a much longer average expression period, are excluded from this estimate. The ICRP has not explicitly projected the risk to miners beyond the years of observation, even though most of the miners on whom their estimates are based are still alive and continuing to die of lung cancer.

The smaller of the two ICRP estimates listed in Table 8.4-3 is based on their dosimetric approach. The ICRP assumes that the risk per rad for lung tissue is 0.12 of the risk of cancer and genetic damage following whole-body exposure (ICRP77). For the case of exposure to radon progeny, the ICRP divided this factor of 0.12 into two equal parts. A weighting factor of 0.06 was used to assess the risk from the high dose to bronchial tissue, where radiogenic lung cancer is observed in exposed underground miners. The other half of the lung weighting factor, another 0.06 of the total body risk, was used to assess the risk to the pulmonary region, which receives a comparatively small dose from radon-222 progeny and where human lung cancer is seldom, if ever, observed.

The UNSCEAR estimate is for a general population and assumes an expression time of 40 years. Like the ICRP, UNSCEAR did not make use of an explicit projection of risk of fatal lung cancer over a full lifetime.

The last entry in Table 8.4-3, the NCRP risk estimate based on an analysis by Harley and Pasternack (USRPC80, Hab82), is of particular interest because, like the EPA and AECB risk estimates, it is based on a life table analysis of the lifetime risk resulting from lifetime exposure. This estimate utilizes an absolute risk projection model with a relatively low risk coefficient, 10 cases per 10^6 person WLM per year at risk, the smallest of those listed by the NAS BEIR-3 Committee, cf. Table 8.4-2. Moreover, they have assumed that the risk of lung cancer following irradiation decreases exponentially with a 20-year half-life so that exposures occurring early in life are of very little risk. The NCRP assumption of a 20-year half-life for radiation injury reduces the estimated lifetime risk by about a factor of 2.5. Without this assumption, the NCRP risk estimate would be the same as the midpoint of the UNSCEAR estimate about 325 fatalities per million person WLM. We find this assumption particularly troublesome. If lung cancer risk decreased over time with a 20-year half-life, the excess lung cancer observed in Japanese A-bomb survivors would have decreased during the period they have been followed, 1950 to 1982. During this period their absolute lung cancer risk has markedly increased (Kab82).

Table 8.4-3 clearly indicates the wide divergence in risk estimates for exposure to radon progeny. In such cases, use of a single risk coefficient may indicate to some that this risk is well known when this obviously not the case. The EPA and AECB estimates may be high because they are relative risk estimates based on males, many of whom smoked. The actual risk to a population which includes women and nonsmokers may be smaller, but it is unlikely to be as small as estimated using the NCRP model. Therefore, on the basis of the BEIR-3, EPA, NRPB, UNSCEAR, and ICRP analyses, risk estimates between 700 and 300 fatalities per million person WLM are reasonable estimates for the possible range of effects resulting from inhaling radon progeny for a full lifetime of exposure. These two risk estimates do not encompass the full range of uncertainty, but do seem to illustrate the breadth of much of current scientific opinion.

8.5 Uncertainties in Risk Estimates for Radiogenic Cancer

As pointed out in the introduction of this chapter, numerical estimates of risks resulting from radiation are neither accurate nor precise. A numerical evaluation of radiogenic cancer risks depends both on epidemiological observations and a number of *ad hoc* assumptions that are largely external to the observed data set. These assumptions include such factors as the expected duration of risk expression and variations in radiosensitivity as a function of age and demographic characteristics. A major assumption is the shape and slope of the dose effects response curve, particularly at low doses where there are little or no epidemiological data. In 1972, the BEIR Committee based its estimates of cancer risk on the assumption that effects at low doses are directly proportional to those observed at high doses, the so called linear-nonthreshold hypothesis. As described above in 8.2, the BEIR-3 Committee considered three dose response models and indicated a preference for the linear quadratic model. The risk coefficients that the BEIR-3 Committee derived for their linear quadratic model, and to a lesser extent for their linear model, are subject to considerable uncertainty primarily because of two factors: (1) systematic errors in the estimated doses of the individual A-bomb survivors and (2) statistical uncertainty because of the small number of cancers observed at various dose levels.

8.5.1 Uncertainty in the Dose Response Models Resulting from Bias in the A-bomb Dosimetry

Although the BEIR-3 Committee's choice of a linear-quadratic response has gained considerable attention, it may not be generally realized that the BEIR-3 Commmittee's numerical evaluations of dose response functions for cancer resulting from low-LET radiation were based exclusively on the cancer mortality of the A-bomb survivors. Unfortunately, the dosimetry for A-bomb survivors, on which the BEIR-3 Committee relied, has since been shown to have large systematic errors that undermine the analyses made by the Committee. As outlined below, the mathematical analyses made by the Committee were "constrained" to meet certain *a priori* assumptions. These assumptions have since been shown to be doubtful.

A careful state-of-the-art evaluation of the dose to A-bomb survivors was carried out by investigators from Oak Ridge National Laboratory in the early 1960s (Aua67, Aub77). These studies resulted in a "T65" dose being assigned to the dose (kerma) in free air at the location of each survivor for both gamma rays and neutrons. A major conclusion of the ORNL study (Aua67, Aub77) was that the mix of gamma ray and neutron radiations was quite different in the two cities where A-bombing occurred. These results indicated that at Hiroshima the neutron dose was more important than the gamma dose when the greater biological efficiency of the high-LET radiations produced by neutrons was taken into account. Conversely, the neutron dose at Nagasaki was shown to be negligible compared to the gamma dose for that range of doses where there were a significant number of survivors. Therefore, the 1980 BEIR Committee evaluated the cancer risks to the survivors at Hiroshima on the assumption that the combined effects of gamma rays and particularly neutrons caused the observed cancer response.

Since the BEIR-3 report was published, it has become evident that the organ doses resulting from neutrons at Hiroshima were overestimated by about an order of magnitude, at distances where most of the irradiated persons survived the bomb blast and yet received significant doses, 1000-1500 meters. In fact, the neutron doses at Hiroshima are quite comparable to those previously assigned, at similar distances, to Nagasaki survivors (Keb81a,b; RERF83,84). Moreover, there are now grounds to believe that the T65 estimates of gamma ray doses in both cities are also incorrect (RERF83,84). Although several factors need further evaluation, reduction of the gamma dose to individual survivors because of the local shielding provided by surrounding structures is significant. The important point, however, is that the overestimate of the neutron dose to the Hiroshima survivors led to the BEIR-3 Committee attributing most of the risk to neutrons rather than gamma-rays. Hence, they underestimated the risk for low-LET radiations by an as yet unknown amount.

For their analysis of the A-bomb survivor data, the BEIR-3 Committee expanded the equations for low-LET radiations listed above in 8.2 to include a linear dose response function for neutrons:

$$P(d,D) = c_1 d + k_1 D \qquad (8\text{-}2)$$

$$P(d,D) = c_2 d^2 + k_2 D \qquad (8\text{-}3)$$

$$P(d,D) = c_3 d + c_4 d^2 + k_3 D \qquad (8\text{-}4)$$

where d is the gamma dose and D is that part of dose resulting from high-LET radiations from neutron interactions. Note that in equation (8-4) the linear-quadratic (LQ) response has two linear terms, one for neutrons and one for gamma radiation. In analyzing approximately linear data in terms of equation (8-4), the decision as to how much of the observed linearity should be assigned to the neutron or the gamma component, i.e,, k_3 and c_3 respectively, is crucial. As shown below, the BEIR-3 Committee attributed most of the observed radiogenic cancer to a linear response from neutron doses that did not occur.

The BEIR-3 Committee's general plan was to examine the dose response for leukemia and for solid cancer separately to find statistically valid estimates of the coefficients $c_1 \ldots\ldots c_4$ and $k_1 \ldots\ldots k_3$ by means of regression analyses. The regressions were made after the data were weighted in proportion to their statistical reliability; thus, Hiroshima results dominate the analysis. The T65 neutron and gamma doses to individual survivors are highly correlated because both are strongly decreasing functions of distance. This makes accurate determination of the coefficients in equation (8-4) by means of a regression analysis extremely difficult. In addition, there is considerable sampling variation in the A-bomb survivor data because of small sample size, which exacerbates the regression problem. Herbert gives a rigorous discussion of these problems for the case of the A-bomb survivors (He83). On account of these and other problems, agreement between the observed response for solid cancers and that predicted by any of the dose response functions examined by the BEIR-3 Committee is not impressive. For example, goodness of fit, based on Chi square, ranges from 0.20 for equation (8-3) to 0.23 for equation (8-4), to 0.30 for equation (8-2) (Table V-11).* For leukemia, the goodness of fit between the observed data and those predicted by the regression analysis is better, e.g. 0.49 for equations (8-2) and (8-3) (Table V8 in NAS80).

The Committee analyzed the A-bomb survivor data in two separate sets, i.e. first leukemia and then all cancer excluding leukemia (solid cancers). Their treatment of these two cases was not equivalent. Unlike the analysis of solid cancers, the Committee's analysis of leukemia considered the Nagaski and Hiroshima data separately. Their approach (p. 342 in NAS80) appears to be based on an unpublished paper by Charles Land and a published report by Ishimaru et al., on estimating the RBE of neutrons by comparing leukemia mortality in Hiroshima to that in Nagasaki (Is79). Unlike the case for solid cancers, see below, the Committee's regression analysis of the leukemia mortality data did provide stable values for all of the coefficients in equation (8-4), and therefore an RBE for neutrons as a function of dose, as well as the ratio of the linear to the dose squared terms for leukemia induction caused by gamma rays, (c_3/c_4).

Estimating the linear-quadratic response coefficients for solid cancers proved to be less straightforward. When the BEIR-3 analysis attempted to fit the A-bomb survivor data on solid cancers to a linear-quadratic dose response function, they found that the linear response coefficient, c_3 in equation (8-4), varied from zero to 5.6 depending on the dose range considered. Moreover, their best estimate of the coefficient for the dose squared term in equation (8-4), i.e., c_4 was zero, i.e., the best fit yielded a linear response. Therefore, it was decided that the observations on solid cancers were "not strong enough to provide stable estimates of low-dose, low-LET radiation cancer risk when analyzed in this fashion" (NAS80, p. 186).

As outlined in the BEIR-3 Report. the Committee decided to use a constrained regression analysis, that is, substitute some of the parameters for equation (8-4) found in their analysis of leukemia deaths to the regression analysis of the dose response for solid cancers. That is, both the neutron RBE at low dose (the ratio of the coefficient k_3 to

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*All references to tables with a V prefix are from Chapter V in NAS80.

c_3) and the ratio of c_3 to c_4 as estimated from the leukemia data were assumed to apply to the induction of fatal solid cancers. Regression analyses that are constrained in this manner can yield much higher estimates of precision than are warranted by the data, as discussed by Land and Pierce (Lac83). They can also be very misleading. Herbert has discussed this point in detail as it applies to the BEIR-3 regression analysis (He83). The BEIR-3 Committee's substitution of the results of the leukemia regression for the data on solid cancers allowed them to make stable estimates of c_3, c_4, and k_3. These estimates became the basis for the "preferred" linear quadratic risk estimates for solid cancers presented in NAS80, i.e., the $\overline{\text{LQ-L}}$ model* (NAS80, p. 187).

Given the information discussed above, it is possible to see, at least qualitatively, how the high bias in the estimated T65 neutron dose to the Japanese survivors affects the 1980 BEIR Committee's "preferred" LQ estimates of the risk coefficients for leukemia. The Committee's age-adjusted risk coefficients for leukemia are listed in Table V-8. For the linear-quadratic response, k_3, the neutron risk coefficient is 27.5. Tables A-11 and V-6 provide the estimates of neutron and gamma doses to the bone marrow of Hiroshima survivors that were used by the Committee. Substituting these doses in their risk equations (Table V-8) indicates that about 70 percent of the leukemia deaths were ascribed to the neutron dose component then thought to be present at Hiroshima. As noted above, subsequent research indicates that the high-LET dose caused by neutrons was actually much smaller.

It is not possible to accurately quantify what effect the Committee's use of these same coefficients had on their analysis of the dose response for solid cancers. Equation V-10 for solid cancers (NAS80, p. 187) indicates that about 60 percent of the solid tumor response was attributed to the T65 neutron dose; but this is a minimum estimate that ignores the effect of the assumed neutron doses on the value of k_3 and the ratio of c_3 to c_4.

The BEIR-3 Committee's $\overline{\text{LQ-L}}$ model assumes an RBE of 27.8 at low doses. In the Committee's $\overline{\text{L-L}}$ linear response model, the assumed RBE is 11.3. Therefore, this linear model is considerably less sensitive to the neutron dose component, assumed by the Committee, than their $\overline{\text{LQ-L}}$ model. For either model, most of the A-bomb survivors' radiogenic cancer was ascribed to the T65 neutron doses at Hiroshima.

There is no simple way of adjusting the 1980 BEIR risk estimates to account for the risk they attributed to neutrons. Adjustment of neutron doses alone is clearly inappropriate, because there is good reason to believe that T65 estimates of the dose caused by gamma rays are also subject to considerable change. Moreover, not all of the individuals in a given T65 dose category will, necessarily, remain grouped together after new estimates of neutron and gamma doses are obtained. Both the numerator and denominator in the ratio of observed to expected cases are subject to change, and indeed could change in opposite directions, a fact not considered in some preliminary (and premature) analyses (Stc81). Nevertheless, it is reasonable to conclude that bias in the estimated neutron doses at Hiroshima has not only led to considerable uncertainty in the BEIR-3 risk estimates but has also led to a systematic underestimation of the risk resulting from low-LET radiations. For this reason we believe that estimates based on the more conservative linear dose response should be given considerable weight *vis-a-vis* those made using the BEIR-3 linear quadratic models.

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*The response models for solid cancers that are based on the Committee's constrained regression analysis are designated with a bar in their 1980 report, e.g., $\overline{\text{LQ-L}}$ and $\overline{\text{L-L}}$.

8.5.2 Sampling Variation

In addition to the systematic bias in the BEIR-3 risk estimates for low-LET radiation outlined above, the precision of the estimated linear and quadratic risk coefficients in the BEIR-3 report is poor as a result of statistical fluctuations caused by sample size. Recently Land and Pierce (Lac83) have reevaluated the precision of the BEIR-3 linear quadratic risk estimates to take account, at least partially, of the Committee's use of a constrained regression analysis. This new analysis indicates that for the BEIR-3 LQ-L model for leukemia, the standard deviation of the linear term is nearly as large as the risk coefficient itself (±0.93 compared to a risk coefficient of 0.99). For the LQ-L model, solid cancer, the standard deviation is ±1.5 compared to a risk coefficient of 1.6.

It is likely that at least part of the uncertainty attributed to sampling variation in the BEIR-3 risk estimates is caused not by sample size and other factors leading to random error but rather by the use of incorrect dose estimates for the A-bomb survivors. The correlation of neutron and gamma-ray doses has been a major underlying cause of the uncertainty in regression analysis using the T65 doses. Analyses of revised data with much smaller neutron doses may result in better precision. At present, we have concluded that the BEIR-3 risk coefficients are uncertain by at least a factor of two, see below, as well as being biased low by an additional factor of two or more.

8.5.3 Uncertainties Arising from Model Selection

In addition to a dose response model, a "transportation model" is needed to apply the risks from an observed irradiated group to another population having different demographic characteristics. A typical example is the application of the Japanese data for A-bomb survivors to western people. Seymore Jablon (Director of the Medical Follow-up Agency of the National Research Council, NAS) has called this the "transportation problem," a helpful designation because it is often confused with the risk projection problem described below. However, there is more than a geographic aspect to demographic characteristics. The "transportation problem" includes estimating the risks for one sex based on data for another and a consideration of habits influencing health status such as differences between smokers and nonsmokers, as described in 8.4 for the case of risk estimates for radon progeny.

The BEIR-3 Committee addressed this problem in their 1980 report and concluded, based largely on the breast cancer evidence, that the appropriate way to transport the Japanese risk to the U.S. population was to assume that the absolute risk over a given observation period was transferrable but that relative risk was not. Therefore, the Committee calculated what the relative risk would be if the same number of excess cancer deaths were observed in a U.S. population having the same age characteristics as the A-bomb survivors. The baseline cancer rates in the U.S. and Japan are quite different for some specific cancers, so this is a reasonable approach. However it contains the assumption that while the cancer initiation process is the same in the two countries, the actual number of radiogenic cancers that actually occur is the result of cancer promotion, the latter being a culturally dependent variable.

An alternative approach to solving the "transportation problem" is that of the 1972 NAS BEIR-1 Committee. This Committee assumed that relative risks would be the same in the U.S. and Japan and transferred the observed percentage increase directly to the U.S. population. We have compared estimates of the lifetime risk for these two treatments of the transportation problem in order to find out how sensitive the BEIR-3 Committee risk estimates are to their assumptions. To do this, we calculated new relative risk estimates for solid cancers based on the age-specific cancer mortality of the Japanese population rather than the U.S. data used by the BEIR-3 Committee. We found that this alternative approach did not have much effect on the estimated lifetime risk of solid radiogenic cancer, i.e. a change of 3 percent for males and 17 percent for females. We have concluded that the amount of uncertainty introduced by transporting cancer risks observed in Japan to the U.S. population is small compared to other sources of uncertainty in this risk assessment. Baseline leukemia rates are about the same in the countries, so we believe that these risks are also "transportable."

The last of the models needed to estimate risk is a risk projection model. As outlined in Section 8.2, such models are used to project what future risks will be as an exposed population ages. For leukemia and bone cancer, where the expression time is not for a full lifetime but rather 25 years, absolute and relative risk projection models yield the same number of radiogenic cancers, but would distribute them somewhat differently by age. For solid cancers, other than bone, the BEIR-3 Committee assumed that radiogenic cancers would occur throughout the lifetime. This makes the choice of projection model more critical, because the relative risk projection yielding estimated risks about three times larger than those obtained with an absolute risk projection, as shown in Table 8.2-2. Because we have used the average of these two projections for solid cancers, we believe reduces the uncertainty resulting from the choice of model to about a factor of two or perhaps less, depending on the age distribution of fatal radiogenic cancer, as outlined in 8.2 above.

Similiarly, there is, as yet, insufficient information of radiosensitivity as function of the age at exposure. The age-dependent risk coefficients that we have used are those presented in the BEIR-3 report. As yet, there is little information on the ultimate effects of exposure during childhood. As the A-bomb survivors' population ages, more information will become available on the cancer mortality of persons irradiated when they were young. Table 8.2-2 indicates that the conservative BEIR-1 estimates for the effect of childhood exposures would increase BEIR-3 risk estimates by about 40 percent. As this is probably an upper limit, the lack of more precise information is not a major source of uncertainty in estimates of the risk caused by lifetime exposure. Similiarly, the BEIR-3 Committee did not calculate population risks for radiogenic cancer that included *in utero* radiation because they felt the available data were unreliable. We have deferred to their judgement in this regard. The BEIR-1 report did include *in utero* cancer risk. These had little effect, 1 to 10 percent, on the lifetime risk of cancer from lifetime exposure. An effect this small is not significant relative to other sources of uncertainty in the risk assessment.

8.5.4 Summary

We can only semi-quantitatively estimate the overall uncertainty in the risk per rad for low-LET radiations. We expect that more quantitative estimates of the uncertainty will be possible only after the A-bomb dose reassessment is completed and the A-bomb survivor data reanalyzed on the basis of the new dose estimates. It should be noted, however, that even if all systematic bias is removed from the new dose estimates, there will still be considerable random error in the dose estimate for each survivor. This random error biases the estimated slope of the dose response curve so that it is smaller than the true dose response (Da72, Maa59). The amount of bias introduced depends on the size of the random error in the dose estimates, and their distribution, which are unknown quantities at this stage of the dose reassessment.

The source of uncertainty in risk estimates for low-LET radiations can be ranked as shown in Table 8.5-1.

The estimates of uncertainty in Table 8.5-1 are not wholly comparable and must be interpeted carefully. However, they do have some illustrative value, particularly when ordered in this way. The uncertainty listed for the *slope* of dose response is a minimal value for the BEIR-3 linear quadratic $\overline{\text{LQ}}$ formulation (Lac83) and is only valid insofar as the Committee's assumptions are true. It is based on two standard deviation errors so that the expectation of the error being less than indicated is 95 percent. We do *not* believe that the uncertainty in the BEIR-3 linear estimate, $\overline{\text{L-L}}$, is significantly smaller, cf. Tables V-9 and V-11 in NAS80.

The other uncertainties listed in Table 8.5-1 are quite different, being more in the nature of informed judgements than the result of a statistical analysis. It is doubtful that all radiogenic cancers have the same type of response functions. However, if they were all linear,

Table 8.5-1. A ranking of causes of uncertainty in estimates of the risk of cancer

Source of uncertainty	Degree of uncertainty
Choice of dose response model	±250%[a]
Slope of dose response resulting from sampling variation	±200%[b]
Choice of an average risk projection model	±100%[c]
Choice of transportation model	±20%[d]
A-bomb T65 dosimetry	Plus only, amount known

[a] For choices limited to BEIR-3 linear and linear quadratic models, see 8.2.

[b] Estimate of 2 standard deviations for the BEIR-3 LQ model (Lac83).

[c] Average of relative and absolute projection as described above.

[d] For the total of all cancers, not specific cancers.

as breast cancer and thyroid appear to be, the BEIR-3 linear quadratic response model would underestimate the response by 250 percent. If most cancers have a linear quadratic response, or equivalently, a dose rate reduction factor equal to the difference in slope at low doses between the BEIR-3 linear and linear quadratic models, use of a linear model would overestimate the response by a factor of 2.5. At present, no one knows which response model is most often appropriate. We believe that a factor of 250 percent is a conservative estimate of the uncertainty introduced by the lack of data at low dose rates.

As discussed above, the uncertainty resulting from the choice of an absolute or a relative risk model is about a factor of three. Use of the average risk for these two models reduces the uncertainty in risk projection by more than a factor of two because it is known that a relative risk projection is high for some kinds of cancer and an absolute risk projection is low for others.

The uncertainties listed in Table 8.5-1 are largely independent of each other and therefore unlikely to be correlated in sign. Their root mean square sum is about 300 percent, indicating the expectation that calculated risks would be within a factor of three or so of the true value. This result is overly optimistic because it does not include consideration of the uncertainty introduced by the bias in the A-bomb dosimetry or by the constrained regression analysis used by the BEIR-3 Committee.

8.6 Other Radiation-Induced Health Effects

The earliest report of radiation induced health effects was in 1896 (Mob67), and it dealt with acute effects in skin caused by x-ray exposures. Within the six year period following, 170 radiation related skin damage cases had been reported. Such injury, like many other acute effects, is the result of exposure to hundreds or thousands of rad. Under normal environmental exposure situations, however, such exposure conditions are not possible and therefore will not be considered in assessing the risk to the general population from radionuclide emissions.

Although radiation-induced carcinogenesis was the first delayed health effect reported, radiation-induced genetic changes were reported early, too. In 1927, H. J. Muller reported on X-ray-induced mutations in animals and in 1928 L. J. Stadler reported a similar finding in plants (Ki62). At about the same time, radiation effects on the developing embryo were reported. Case reports in 1929 showed a high rate of microcephaly (small head size) and nervous system disturbance and one case of skeletal defects in children irradiated in utero (UNSCEAR69). These effects, at unrecorded but high exposures, appeared to be central nervous system and eye defects similar to those reported in rats as early as 1922 (Rub50).

For purposes of assessing the risks of environmental exposure to radionuclide emissions, the genetic effects and in utero developmental effects are the only health hazards other than cancer that are addressed in this BID.

8.6.1 Types of Genetic Harm and Duration of Expression

Genetic harm or the genetic effects of radiation exposure are those effects induced in the germ cells (eggs or sperm) of exposed individuals, which are transmitted to and expressed in their progeny and future generations.

Of the possible consequences of radiation exposure, the genetic risk is more subtle than the somatic risks. Genetic risk is incurred by fertile people when radiation damages the nucleus of the cells that become their eggs or sperm. Damage, in the form of a mutation or a chromosome aberration, is transmitted to, and may be expressed in, a child conceived after the radiation exposure and subsequent generations. However, the damage may be expressed only after many generations or, alternately, it may never be expressed because of failure to reproduce.

The EPA treats genetic risk as independent of somatic risk because, although somatic risk is expressed in the person exposed, genetic risk is expressed only in progeny and, in general, over many subsequent generations. Moreover, the types of damage incurred often differ in kind from cancer and cancer death. Historically, research on genetic effects and development of risk estimates has proceeded independently of the research on carcinogenesis. Neither dose response models nor risk estimates used for genetics are derived from data on studies of carcinogenesis.

Although genetic effects may vary greatly in severity, the genetic risks considered by EPA in evaluating the hazard of radiation exposure include only those "disorders and traits that cause a serious handicap at some time during lifetime" (NAS80). Genetic risk may result from one of several types of damage that ionizing radiation can cause in the DNA within eggs and sperm. The types of damage usually considered are: dominant and recessive mutations in autosomal chromosomes, mutations in sex-linked (x-linked) chromosomes, chromosome aberrations (physical rearrangement or removal of part of the genetic message on the chromosome or abnormal numbers of chromosomes), and irregularly inherited disorders (genetic conditions with complex causes, constitutional and degenerative diseases, etc.).

Estimates of the genetic risk per generation are based on a 30 year reproductive generation. That is, the median parental age for production of children is age 30 (one half the children are produced by persons less than age 30, the other half by persons over age 30). Thus, the radiation dose accumulated up to age 30 is used to estimate the genetic risks. Using this accumulated dose and the number of live births in the population along with the estimated genetic risk per unit dose, it is possible to estimate the total number of genetic effects per year, those in the first generation and the total across all time. Most genetic risk analyses have provided such data. EPA assessment of risks of genetic effects includes both first generation estimates and total genetic burden estimates.

Direct and Indirect Methods of Obtaining Risk Coefficients for Genetic Effects

Genetic effects, as noted above, may occur in the offspring of the exposed individuals or they may be spread across all succeeding generations. Two methods have been used to estimate the frequency of mutations in the offspring of exposed persons, direct and indirect. In either case, the starting point is data from animal studies, not data obtained from studies of human populations.

For a direct estimate, the starting point is the frequency of a mutation per unit exposure in some experimental animal study. The 1982 UNSCEAR (UNSCEAR82) report gave an example of the direct method for estimating induction of balanced reciprocal translocations (a type of chromosomal aberration) in males per rad of low level, low-LET radiation.

	Induction Rate/rad
(1) Rate of induction in rhesus monkey spermatogonia: cytogenetic data	0.86×10^{-4}
(2) Rate of induction that relates to recoverable translocations in the F_1 (1st filial generation) progeny [divide (1) by 4]	0.215×10^{-4}
(3) Rate after low dose rate X-rays: based on mouse cytogenetic observations [divide (2) by 2]	0.1075×10^{-4}
(4) Rate after chronic gamma-irradiation: based on mouse cytogenetic observations [divide (2) by 10]	0.022×10^{-4}
(5) Expected rate of unbalanced products [multiply (3) and (4) by 2]	for (3): 0.215×10^{-4} for (4): 0.043×10^{-4}
(6) Expected frequency of congenitally malformed children in the F_1, assuming that about 6 percent of unbalanced products [item (5) above] contribute to this	
for low dose rate X-rays	1.3×10^{-6}
for chronic gamma radiation	$\sim 0.3 \times 10^{-6}$

For humans, UNSCEAR estimates that a consequence of induction of balanced reciprocal translocations in exposed fathers, an estimated 0.3 to 1.3 congenitally malformed children would occur in each 10^6 live births for every rad of parental radiation exposure.

A complete direct estimate of genetic effects would include estimates, derived in a manner similar to that shown above for each type of genetic damage. These direct estimates could be used to calculate the risk of genetic effects in the first generation (F_1) children of exposed parents.

The indirect (or doubling dose) method of estimating genetic risk also uses animal data but in a different way. The 1980 BEIR-3 report (NAS80) demonstrates how such estimates are obtained:

	Induction Rate/rad
(1) Average radiation-induced mutation per gene for both sexes in mice [based on 12 locus data in male mice]: induction rate per rad	0.25×10^{-7}

(2)	Estimated human spontaneous mutation rate per gene	0.5 x 10^{-6} to 0.5 x 10^{-5}
(3)	Relative mutation risk in humans [divide (1) by (2)]	0.005 to 0.05
(4)	Doubling dose: the exposure needed to double the human mutation rate	200 to 20 rad

The doubling dose can then be used to estimate the equilibrium genetic effects or the genetic burden in all future generations caused by the exposure of parents. Since the genetic component of congenital defects occurring in the population can be estimated by epidemiological surveys, and this component is considered to be maintained at an equilibrium level by mutations, a doubling dose of ionizing radiation would double these genetic effects. Dividing the number of the various genetic efects in 10^6 live births by the doubling dose yields the estimate of genetic effects per rad. For example:

(1)	Autosomal dominant and x-linked diseases, current incidence	10,000 per 10^6 live births
(2)	Estimated doubling dose	20 to 200 rad
(3)	Estimate of induced autosomal dominant and x-linked diseases	50 to 500 per 10^6 live births per rad of parental exposure.

The doubling dose estimate assumes that the total population of both sexes is equally irradiated, as occurs from background radiation, and that the population exposed is large enough so that all genetic damage can be expressed in future offspring. Although it is basically an estimate of the total genetic burden across all future generations, the doubling dose estimate can also estimate the effects that occur in the first generation. Usually a fraction of the total genetic burden for each type of damage is assigned to the first generation using population genetics data as a basis to determine the fraction. For example, the BEIR-3 committee geneticists estimated that one-sixth of the total genetic burden of x-linked mutations would be expressed in the first generation, five-sixths across all future generations. EPA assessment of risks of genetic effects includes both first generation estimates and total genetic burden estimates.

8.6.2 Estimates of Genetic Harm Resulting from Low-LET Radiations

One of the first estimates of genetic risk was made in 1956 by the NAS Committee on the Biological Effects of Atomic Radiation (BEAR Committee). Based on *Drosophila* (fruit fly) data and other considerations, the BEAR Genetics Committee estimated that 10 Roentgens

(10 R*) per generation continued indefinitely would lead to about 5,000 new instances of "tangible inherited defects" per 10^6 births, and about one-tenth of them would occur in the first generation after the irradiation began (NAS72). The UNSCEAR addressed genetic risk in their 1958, 1962, and 1966 reports (UNSCEAR58, UNSCEAR62, UNSCEAR66). During this period, they estimated that one rad of low-LET radiation would cause a 1 to 10 percent increase in the spontaneous incidence of genetic effects.

In 1972, both the NAS BEIR Committee (NAS72) and UNSCEAR (UNSCEAR72) reexamined the question of genetic risks. Although there were no definitive human data, additional information was available on the genetic effects of radiation on mammals and insects. In 1977, UNSCEAR reevaluated the 1972 genetics estimates (UNSCEAR77). Their new estimates used recent information on the current incidence of various genetic conditions, along with additional data on radiation exposure of mice and marmosets and other considerations.

In 1980, an ICRP Task Group (ICRPTG) summarized recommendations that formed the basis for the genetic risk estimates published in ICRP Report 26 (Of80). These risk estimates are based on data similar to those used by the BEIR and UNSCEAR Committees, but used slightly different assumptions and effect categories, Table 8.6-1.

The 1980 NAS BEIR Committee revised genetic risk estimates (NAS80). The revision considered much of the same material that was in BEIR-1 (NAS72), the newer material considered by UNSCEAR in 1977, and some additional data. Estimates for the first generation are about a factor of two smaller than reported in the BEIR-1 report. For all generations, the new estimates are essentially the same, Table 8.6-2.

The most recent genetic risk estimate, in the 1982 UNSCEAR Report (UNSCEAR82), includes some new data on cells in culture and the results of genetic experiments using primates rather than rodents, Table 8.6-3.

Although all the reports described above used somewhat different sources of information, there is reasonable agreement in the estimates (see the summary in Table 8.6-4). Most of the difference is caused by the newer information used in each report. Note that all estimates listed above are based on the extrapolation of animal data to man. Groups differ in their interpretation of how genetic experiments in animals might be expressed in humans. Although there are no comparable human data at present, information on hereditary defects among the children of A-bomb survivors provide a degree of confidence that the animal data do not lead to underestimates of the genetic risk following exposure to humans. (See "Observations on Human Populations" which follows.)

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*R is the symbol for Roentgen, a unit of measurement of x-radiation, equivalent to an absorbed dose in tissue of approximately 0.9 rad.

Table 8.6-1. ICRP task group estimate of number of cases of serious genetic ill health in liveborn from parents irradiated with 10^6 man-rem in a population of constant size[a] (Assumed Doubling Dose = 100 rad)

Category of genetic effect	First generation	Equilibrium
Unbalanced translocations: risk of malformed liveborn	23	30
Trisomics and XO	30	30
Simple dominants and sex-linked mutations	20	100
Dominants of incomplete penetrance and multifactorial disease maintained by mutation	16	160
Multifactorial disease not maintained by mutation	0	0
Recessive disease	--	--
Total	89	320

(a)This is equivalent to effects per 10^6 liveborn following an average parental population exposure of 1 rem per 30-year generation, as used by BEIR and UNSCEAR.

Source: (Of80).

Table 8.6-2. BEIR-3 estimates of genetic effects of an average population exposure of 1 rem per 30-year generation

Type of genetic disorder	Current incidence per 10^6 liveborn	Effects per 10^6 liveborn per rem per generation	
		First Generation	Equilibrium
Autosomal dominant and x-linked	10,000	5-65	40-200
Irregularly inherited	90,000	(not estimated)	20-900
Recessive	1,100	Very few	Very slow increase
Chromosomal aberrations	6,000	Fewer than 10	Increases only slightly
Total	107,100	5-75	60-1100

Source: (NAS80).

Table 8.6-3. UNSCEAR 1982 estimated effect of 1 rad per generation of low dose or low dose rate, low-LET radiation on a population of 10^6 liveborn according to the doubling dose method (Assumed Doubling Dose = 100 rad)

Disease classification	Current incidence	Effect of 1 rad per generation	
		First Generation	Equilibrium
Autosomal dominant and x-linked diseases	10,000	15	100
Recessive diseases	2,500	Slight	slow increase
Chromosomal diseases:			
Structural	400	2.4	4
Numerical	3,000	Probably very small	
Congenital anomalies, anomalies expressed later, constitutional and degenerative diseases	90,000	4.5	45
Total	105,900	22	149

Source: (UNSCEAR82).

Table 8.6.4. Summary of genetic risk estimates per 10^6 liveborn for an average population exposure of 1 rad of low dose or low dose rate, low-LET radiation in a 30-year generation

Source	Serious hereditary effects	
	First generation	Equilibrium (all generations)
BEAR, 1956 (NAS72)	--	500
BEIR-I, 1972 (NAS72)	49[(a)] (12-200)	300[(a)] (60-1500)
UNSCEAR, 1972 (UNSCEAR72)	9[(a)] (6-15)	300
UNSCEAR, 1977 (UNSCEAR77)	63	185
ICRPTG, 1980 (Of80)	89	320
BEIR-3, 1980 (NAS80)	19[(a)] (5-75)	257[(a)] (60-1100)
UNSCEAR, 1982 (UNSCEAR82)	22	149

Numbers in parentheses ( ) are the range of estimates

[(a)] Geometric Mean is calculated by taking the square root of the product of two numbers for which the mean is to be calculated. The cube root of three numbers, etc. In general, it is the Nth root of the product of N numbers for which the mean is to be calculated.

It should be noted that the genetic risk estimates summarized in Table 8.6-4 are for low-LET, low dose, and low dose rate irradiation. Most of the data were obtained from high dose rate studies and most authors have used a sex-averaged factor of 0.3 to correct for the change from high dose rate, low-LET to low dose rate, low-LET exposure (NAS72, NAS80, UNSCEAR72, UNSCEAR77). However, factors of 0.5 to 0.1 have been used in estimates of specific types of genetic damage (UNSCEAR72, UNSCEAR77, UNSCEAR82, Of80).

8.6.3 Estimates of Genetic Harm for High-LET Radiations

Although genetic risk estimates are made for low-LET radiation, some radioactive elements deposited in the ovary or testis can irradiate the germ cells with alpha particles. The ratio of the dose (rad) of low-LET radiation to the dose of high-LET radiation producing the same endpoint is called RBE and is a measure of the effectiveness of high-LET compared to low-LET radiation in causing the same specific endpoint.

Studies in which the beta particle emitted isotopes carbon-14 and tritium yielded RBEs of 1.0 and 0.7 to about 2.0, respectively (UNSCEAR82). At the present time, the RBE for genetic endpoints resulting from beta particles is taken as one (UNSCEAR77, UNSCEAR82).

Studies of the RBE for alpha-emitting elements in germinal tissue have used only plutonium-239. Studies comparing cytogenetic endpoints after chronic low dose rate gamma radiation exposure or incorporation of plutonium-239 in the mouse testis, have yielded RBEs of 23 to 50 for the type of genetic injury (reciprocal translocations) that might be transmitted to liveborn offspring (NAS80, UNSCEAR77, UNSCEAR82). However, an RBE of 4 for plutonium-239 compared to chronic low-LET radiation was reported for specific locus mutations observed in neonate mice (NAS80). Neutron RBE, determined from cytogenetic studies in mice, also ranges from about 4 to 50 (UNSCEAR82, Gra83, Ga82). Most reports use an RBE of 20 to convert risk estimates for low dose rate, low-LET radiation to risk estimates for high-LET radiation.

8.6.4 Uncertainty in Estimates of Radiogenetic Harm

Chromosomal damage and mutations have been demonstrated in cells in culture, in plants, in insects, and in mammals (UNSCEAR72, UNSCEAR77, UNSCEAR82). Chromosome studies in peripheral blood lymphocytes of persons exposed to radiation have shown a dose-related increase in chromosome aberrations (structural damage to chromosome) (UNSCEAR82). In a study of nuclear dockyard workers exposed to external X-radiation at rates of less than 5 rad per year, Evans et al. (Ev79) found a significant increase in the incidence of chromosome aberrations. The increase appeared to have a linear dependence on cumulative dose. In a study of people working and living in a high natural background area where there was both external gamma radiation and internal alpha radiation, Pohl-Ruling et al. (Po78) reported a complex dose response curve. For mainly gamma radiation exposure (less than 10 percent alpha radiation), they reported the the increase in chromosome aberrations increased linearly from 100 to 200 mrad per year, then plateaued from 300 mrad to 2 rad per year. They concluded:

> "From these data, and data in the literature, it can be concluded that the initial part of the dose-effect curve for chromosome aberrations is not linear or sigmoid with a threshold at the lowest dose, but rises sharply and passes into a complex upward form with a kind of plateau until it meets the linear curve of the high dose."

Although chromosomal damage in peripheral blood lymphocytes cannot be used for predicting genetic risk in progeny of an exposed person, it is believed by some to be a direct expression of the damage analogous to that induced in germ cells as a result of the radiation exposure. It is at least evidence that chromosome damage can occur *in vivo* in humans.

Since there are no quantitative human data on genetic risks following radiation exposure, risk estimates are based on extrapolations from animal data. As genetic studies proceeded, emphasis has shifted from *Drosophila* to mammalian species in attempts to find an experimental system that would reasonably project what might happen in humans.

For example, Van Buul (Va80) reported the slope (b) of the linear regression, Y = a + bD, for induction of reciprocal translocations in spermatogonia (one of the stages of sperm development) in various species as follows:

	$b \times 10^4 \pm sd \times 10^4$
Rhesus monkey	0.86 ± 0.04
Mouse	1.29 ± 0.02 to 2.90 ± 0.34
Rabbit	1.48 ± 0.13
Guinea pig	0.91 ± 0.10
Marmoset	7.44 ± 0.95
Human	3.40 ± 0.72

These data indicate that animal based estimates for this type of genetic effect would be within a factor of four of the true human value. In this case most of the animal results would underestimate the risk in humans.

However, when risk estimates such as this are used in direct estimation of risk for the first generation, the total uncertainty in the estimate becomes indeterminate. Even if studies have been made the results which can be used to predict the dose response and risk coefficient for a specific radiation-induced genetic damage for a species, there is no certainty that this prediction will represent the response for all genetic damage of that type. In addition, as shown in the example from the 1982 UNSCEAR report (UNSCEAR82) shown in Section 8.6.1, additional assumptions based on observations, usually in other animal species, are used to adjust the risk coefficient to what is expected for humans. The uncertainty in these extrapolations has not been quantified.

A rough estimate of the uncertainty can be obtained by comparing direct estimates of risk for the first generation with doubling dose estimates in the 1977 UNSCEAR report (UNSCEAR77). The estimates differ by a factor of between 2 and 6 with the direct estimate usually smaller than the doubling dose estimate.

A basic assumption in the doubling dose method of estimation is that there is a proportionality between radiation-induced and spontaneous mutation rates. Some of the uncertainty was removed in the 1982 UNSCEAR report (UNSCEAR82) with the observation that in two test systems (fruit flies and bacteria) there is a proportionality between spontaneous and induced mutation rates at a number of individual gene sites. There is still some question as to whether the sites that have been examined are representative of all sites and all gene loci or not. The doubling dose estimate does, however, seem better supported than the direct estimate.

Although there is still some uncertainty as to what should be doubled, future studies on genetic conditions and diseases can only increase the total number of such conditions. Every report, from the 1972 BEIR and UNSCEAR reports to the most recent, has listed an increased number of conditions and diseases that have a genetic component.

Observations on Human Populations

As noted earlier, the genetic risk estimates are based on interpretation of animal experiments as applied to data on naturally occurring hereditary diseases and defects in man. A study of birth cohorts was initiated in the Japanese A-bomb survivors in mid-1946. This resulted in a detailed monograph by Neel and Schull (Nea56), which outlined the background of the first study and made a detailed analysis of the findings to January 1954 when the study terminated. The authors concluded only that it was improbable that human genes were so sensitive that exposures as low as 3 R, or even 10 R, would double the mutation rate. Although this first study addressed morphological endpoints, subsequent studies have addressed other endpoints. The most recent reports on this birth cohort of 70,082 persons have attempted only to estimate the minimum doubling dose for genetic effects in man (Sc81, Sa82).

Data on four endpoints have been recorded for this birth cohort. Frequency of stillbirths, major congenital defects, perinatal death, and frequency of death prior to age 17, have been examined in the entire cohort. Frequency of cytogenetic aberrations (sex chromosome aneuploidy) and frequency of biochemical variants (a variant enzyme or protein electrophoresis pattern) have been measured on large subsets of this cohort.

Although the updated data reported appear to suggest that radiation effects have occurred, the numbers are small and not statistically significant. Overall, the estimated doubling dose for low-LET radiation at high doses and dose rates for human genetic effects is about 156 rem (Sc81) or 250 rem (Sa82). As noted above, animal studies indicate that chronic exposures to low-LET radiation would be less hazardous by a factor of three (NAS72, 80). This would increase the estimated doubling dose to 468 to 750 rem, respectively. These recent reports suggest that the minimum doubling dose for humans may be from 4 to 7 times higher than those in Table 8.6-4 (based on animal data). It would be premature to reach a firm decision on this point, because these reports are based on the T65D dosimetry in Japan,* which is being revised. However, we believe EPA estimates of genetic risks will prove to be conservative even when the dosimetry of A-bomb survivors is revised.

EPA is using the geometric mean of the BEIR-3 range of doubling doses, about 110 rad. The minimum doubling dose reported above is 4 to 7 times greater. It is unlikely that dose estimates for Japanese survivors will change by this much (RERF83, 84). Therefore, EPA believes the estimate of a doubling dose of about 100 rad will continue to be a conservative estimate.

Ranges of Estimates Provided by Various Models

EPA has continued to follow the recommendations of the 1980 BEIR-3 committee and use a linear nonthreshold model for estimating genetic effects. As pointed out by the 1982 UNSCEAR committee, there are a number of models other than linear ($Y = c + aD$)**--e.g., linear quadratic ($Y = c + bD + eD^2$), quadratic ($Y = k + fD^2$), even power function ($Y = k + gD^h$). However, there are strong data to support the hypothesis that mutations themselves are single track events. That is, the mutations follow a linear dose response function, while the observed mutation rate shows the influence of other factors and may be nonlinear (UNSCEAR82).

Most arguments for a nonlinear dose response have been based on target theory (Le62) or microdosimetric site theory (Kea72). However, other theories based on biology (e.g. enzyme induction-saturation (Gob80, 82), repair-misrepair (Toa80), etc.) could also provide models that fit the observed data. There is still much disagreement on which dose response model is appropriate for estimating genetic effects in humans. Until there is more consensus the linear nonthreshold model appears to be a prudent approach that will not grossly underestimate the risks.

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*See Section 8.2.

**Y is yield of genetic effects;D is radiation dose;c, C, k, and K are spontaneous incidence constants for genetic effects; and a, b, e, f, g, and h are the rate constants for radiation induced genetic effects.

The agreement in estimates made on a linear nonthreshold model, in the various reports, is quite good. Even though the authors of the reports used different animal models, interpreted them in different ways, and gave different estimates of the level of human genetic conditions in the population, the range is about an order of magnitude (see Table 8.6-4). For the most recent more comparable estimates, the range is a factor of two to four (see ICRPTG, BEIR-3 and UNSCEAR 1982 in Table 8.6-4).

8.6.5 The EPA Genetic Risk Estimate

There is no compelling evidence for preferring any one set of the genetic risk estimates listed in Table 8.6-4. EPA has used the estimates from BEIR-3 (NAS80). These "indirect" estimates are calculated using the normal prevalence of genetic defects and the dose that is considered to double this risk. The NAS estimates used by EPA are based on a doubling dose range, with a lower bound of 50 rem and an upper bound of 250 rem. We prefer these risk estimates to those made by the ICRP task group (Of80), which used a "direct" estimate, because the ICRPTG tabulation combines "direct" estimates for some types of genetic damage with doubling dose estimates for others. We also prefer the BEIR-3 risk estimates to the "direct" estimates of UNSCEAR 1982, which tabulates genetic risk separately by the direct method and by the doubling dose method. The risk estimated by the direct method does not include the same types of damage estimated by doubling doses and was not considered further. The BEIR-3 genetic risk estimate is also preferred over the UNSCEAR 1982 and ICRPTG estimates, because BEIR-3 assigns a range of uncertainty for multifactorial diseases (>5 percent to <50 percent) which reflects the uncertainty in the numbers better than the other estimates do (5 percent and 10 percent, respectively).

In developing the average mutation rate for the two sexes used in the calculation of the relative mutation risk, the BEIR-3 committee postulated that the induced mutation rate in females was about 40 percent of that in males (NAS80). Recent studies by Dobson et al. (Doa83, Dob83, Doc84, Dod84) suggest that the assumption was invalid and that human oocytes should have a risk equivalent to that of human spermatogonia. This would increase the risk estimate obtained by doubling-dose methods by a factor of 1.43.

We recognize, however, that the use of the doubling dose concept does assume that radiation-induced genetic damage is in some way proportional to "spontaneous" damage. As noted earlier, the recent evidence obtained in insects (Drosophila) and bacteria (E. coli) supports the hypothesis that, with the exception of "hot spots" for mutation, the radiation-induced mutation rate is proportional to the spontaneous rate (UNSCEAR82). No proof that this is also true in mammals is available yet.

The BEIR-3 estimates give a considerable range. To express the range as a single estimate, the geometric mean of the range is used, a method first recommended by UNSCEAR (UNSCEAR58) for purposes of calculating genetic risk. The factor of three increase in risk for high dose rate, low-LET radiation noted earlier is also used.

The question of RBE for high-LET radiation is more difficult. As noted above, estimated RBEs for plutonium-239 alphas versus chronic gamma radiation for reciprocal translocations as determined by cytogenetic analyses is between 23 and 50 (NAS80, UNSCEAR82). However, the observed RBE for single locus mutations in developing offspring of male mice given plutonium-239 compared to those given X-ray irradiation is 4 (NAS80). The average of RBEs for reciprocal translocations and for specific locus mutations is 20.25. Since reported neutrons RBEs are similar to those listed above for plutonium-239 alpha radiation, we use an RBE of 20 to estimate genetic risks for all high-LET radiations. This is consistent with the RBE for high-LET particles recommended for estimated genetic risks associated with space flight (Grb83).

Genetic risk estimates used by EPA for high- and low-LET radiations are listed in Table 8.6-5. As noted above, EPA uses the dose received before age 30 in assessing genetic risks.

The EPA estimates (Table 8.6-5), like all other human genetic risk estimates, are limited by the lack of confirming evidence of genetic effects in humans. These estimates depend on a presumed resemblance of radiation effects in animals. The magnitude of the possible error is

Table 8.6-5. EPA estimated frequency of genetic disorders in a birth cohort due to exposure of the parents to 1 rad per generation

Type of radiation	Cases per 10^6 liveborn			
	First generation		All generations	
	low[a]	high[b]	low[a]	High[b]
Low dose rate, low-LET	20	30	260	370
High dose rate, low-LET	60	90	780	1110
High-LET	400	600	5200	7400

[a] Female sensitivity to induction of genetic effects is 40 percent as great as that of males.

[b] Female sensitivity to induction of genetic effects is equal to that of males.

indeterminable. The study with the largest data base, that of the Japanese A-bomb survivors, appears at best to provide an estimate of the minimum doubling dose for calculating the maximum genetic risk in man. However, doubling dose estimates are also uncertain because the number of human disorders having a recognized genetic component is constantly increasing, and the type of genetic damage implicated in a specific disorder may change. The combined uncertainties in doubling dose estimates and the magnitude of genetic contributions to various disorders probably introduces an overall uncertainty of about an order of magnitude in the risk estimates. Moreover, the BEIR Committee in deriving its estimates has assumed that almost all of the risk was caused by recessive mutations that would eventually be eliminated. To what extent this occurs will depend on medical practices in the future. It is possible, as our knowledge of medicine improves, that recessive hereditary defects will be carried on for many more generations than assumed by the BEIR Committee.

The relative risk of high-LET radiation compared to low dose rate, low-LET radiation (RBE) is also uncertain. The data are sparse, and different studies often used different endpoints. In addition, the microscopic dosimetry, i.e., the actual absorbed dose in the cells at risk, is poorly known. However, the RBE estimate used by EPA should be within a factor of five of the true RBE for high-LET radiation.

8.6.6 Teratogenic Effects

Although human teratogenesis (congenital abnormalities or defects) associated with X-ray exposure has a long history, the early literature deals only with case reports. Stettner reported a case in 1921 (Stb21) and Murphy and Goldstein studied a series of pregnancies in which 18 of the children born to 76 irradiated mothers were microcephalic (Mub29, Goa29). However, the irradiation exposures were high.

In 1930, Murphy exposed some rats to X-rays at doses of 200 R to 1600 R. Thirty-four of 120 exposed females had litters and five of the litters had animals with developmental defects (Muc30). He felt that this study confirmed his clinical observations and earlier reports of animal studies. Although there were additional studies of radiation-induced mammalian teratogenesis before 1950, the majority of the studies were conducted after that time (see Ru53 for a review), perhaps reflecting the radiological hazards caused by the explosion of the first nuclear weapons in 1945 (Jab70).

Much of the work done after the second world war was done in mice (Rub50, Ruc54, Rub56) and rats (Wi54, Hib54). Early studies, at relatively high radiation exposures, 25 R and above, established some dose response relationships. More importantly, they established the timetable of sensitivity of the developing rodent embryo and fetus to radiation effects (Ruc54, Hia53, Se69, Hic66).

Rugh, in his review of radiation teratogenesis (Rua70), listed the reported mammalian anomalies and the exposure causing them. The lowest reported exposure was 12.5 R for structural defects and 1 R for functional defects. He also suggested that human exposure between ovulation and about 7 weeks gestational age could lead to structural defects and from about 6 weeks gestational age till birth could lead to functional defects. In a later review (Rua71), he suggested that structural defects in the skeleton might be induced as late as the 10th week of gestation and functional defects as early as the 4th week. It should be noted that the gestation period in mice is much shorter than for humans and that weeks of gestation referred to above are in terms of equivalent stages of mouse-human development. Estimates of equivalent gestational age are not very accurate.

In the reports of animal studies it appeared as if teratologic effects, other than perhaps growth retardation, had a threshold for induction of effects (Ruc54, Rua53, Wi54). However, Ohzu (Oh65) showed that doses as low as 5 R to preimplantation mouse embryos caused increased resorption of implanted embryos and structural abnormalities in survivors. Then in 1970, Jacobsen (Jab70) reported a study in which mice were exposed to 5, 20 or 100 R on the 8th day of pregnancy. He concluded that the dose-response function for induction of skeletal effects was linear, or nearly linear, with no observable threshold. This appeared consistent with a report by Russell (Rub57), which suggested a threshold for some effects whereas others appeared linear.

Rugh (Rua71) suggested there may be no threshold for radiation induced congenital effects in the early human fetus. In the case of microcephaly (small head size) and mental retardation at least this may be the case. For other teratogenic effects the dose response in humans is unknown. In 1978, Michel and Fritz-Niggli (Mi78) reported induction of a significant increase in growth retardation, eye and nervous system abnormalities, and post implantation losses in mice exposed to 1 R. The increase was still greater if there was concurrent exposure to radiosensitizing chemicals such as iodoacetimide or tetracycline (Mi78).

One of the problems with the teratologic studies in animals is the difficulty of determining how dose response data should be interpreted.

Russell (Ruc54) pointed out some aspects of the problem: (1) although radiation is absorbed throughout the embryo, it causes selective damage which is consistently dependent on the stage of embryonic development at the time of irradiation and (2) the damaged parts respond, in a consistent manner, within a narrow time range. However, while low dose irradiation at a certain stage of development produces changes only in components at their peak sensitivity, higher doses may induce additional abnormalities which have peak sensitivity at other stages of development, and may further modify expression of the changes induced in parts of the embryo at peak sensitivity during the time of irradiation. In the first case, damage may be to primordial cells themselves, while in the second, the damage may lead indirectly to the same or different endpoints.

The embryo*/fetus** starts as a single fertilized egg and divides and differentiates to produce the normal infant at term. The different organ and tissue primordia develop independently and at different rates. However, they are in contact through chemical induction or evocation (Arb54). These chemical messages between cells are important in bringing about orderly development and the correct timing and fitting together of parts of organs or organisms. While radiation can disrupt this pattern, interpretation of the response may be difficult. Since the cells in the embryo/fetus differentiate, divide and proliferate at different times during gestation and at different rates, gestional times when cells of specific organs or tissues reach maximum sensitivity to radiation are different. Each embryo/fetus has a different timetable. In fact, each half (left/right) of an embryo/fetus may have a slightly different timetable.

In addition, there is a continuum of variation from the hypothetical normal to the extreme deviant, which is obviously recognizable. There is no logical place to draw a line of separation between normal and abnormal. The distinction between minor variations of normal and frank malformation, therefore, is an arbitrary one and each investigator must establish his own criteria and apply them to spontaneous and induced abnormalities alike (HWC73). For example, some classify mental retardation as IQ 80 or lower, some classify on ability to converse or hold a job, some on the basis of the need to be institutionalized.

Because of the problems in interpretation listed above, it appears a pragmatic approach is useful. The dose response should be given as the simplest function that fits the data, often linear or linear with a threshold. No attempt should be made to develop complex dose response models unless the evidence is unequivocal.

The first report of congenital abnormalities in children exposed in utero to radiation from atomic bombs was that of Plummer (P152). Twelve children with microcephaly of which 10 also had mental retardation had been identified in Hiroshima in the _in utero_ exposed survivors. They were found as part of a program started in 1950 to study children exposed in the first trimester of gestation. In 1955 the program was expanded to include all survivors exposed _in utero_.

Studies initiated during the program have shown the following radiation-related effects: (1) growth retardation; (2) increased microcephaly; (3) increased mortality, especially infant mortality; (4) temporary suppression of antibody production against influenza; and

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*The embryonic period, when organs develop, is the period from conception to 7 weeks gestational age.

**The fetal period, a time of _in utero_ growth, is the period from 8 weeks gestational age to birth.

(5) increased frequency of chromosomal aberrations in peripheral lymphocytes (Kaa73).

Although there have been a number of studies of Japanese A-bomb survivors, including one showing a dose and gestational age related increase in postnatal mortality (Kaa73), only incidences of microcephaly and mental retardation have been investigated in any great detail. In the most recent report, Otake and Schull (Ot83) showed that mental retardation was associated with exposure between 8 and 15 weeks of gestation (10 to 17 weeks of gestation if counted from the last menstrual period). They further found a linear dose-response relationship for induction of mental retardation that had a slope yielding a doubling dose for mental retardation of about 2 rad, fetal-absorbed dose (Ot83). Classification as mentally retarded was based on "unable to perform simple calculations, to care for himself or herself, or if he or she was completely unmanageable or had been institutionalized". (Ot83)

Estimates of the risk of mental retardation for a rad of embryo/fetus exposure in the U.S. population can be derived by three methods. The first and easiest method is to use the absolute risk calculated by Otake and Schull for the Japanese survivors (Ot84). A second method is to use the doubling dose calculated by Otake and Schull (Ot83) times the incidence of mental retardation per 10^3 live births. Unfortunately, a number of assumptions must be made to establish the incidence of mental retardation per 10^3 live births. Mental retardation may be classified as mild (IQ 50-70), moderate (IQ 35-49), severe (IQ 20-34) and profound (IQ <20) (WHO75). However, some investigators use only mild mental retardation (IQ 50-70) and severe mental retardation (IQ <50) as classes (Haa81, Sta84). Mental retardation is not usually diagnosed at birth but at some later time, often at school age. Since the mental retardation may have been caused before or during gestation, at the time of birth or at some time after birth, that fraction was caused before or during gestation must be estimated. In like manner since mental retardation caused before birth may be due to genetic conditions, infections, physiologic conditions, etc.; the fraction related to unknown causes during gestation must be estimated. This is the fraction that might possibly be doubled by radiation exposure.

A third method to estimate the risk is indirectly, using the relationship of microcephaly and mental retardation reported in the Japanese survivors (Woa65, Ot83). If head size is assumed to be normally distributed, then the fraction of the population with a head size 2 or 3 standard deviations smaller than average can be obtained from statistical tables. The fraction of 10^3 liveborn with microcephaly multiplied by the proportion of mental retardation associated with that head size yields an estimate of the incidence of mental retardation per 10^3 live births; which can then be used with the doubling dose to estimate the risk as described above.

Risk estimates for mental retardation are derived below for comparison purposes using each of the three methods described above.

A. Estimate of Incidence Per Rad Based on Direct Application of the Slope of the Japanese Data

Otake and Schull (Ot84) gave an estimate of 'The Relationship of Mental Retardation to Absorbed Fetal Exposure in the "Sensitive" Period when All "Controls" are Combined.' The estimate of 0.416 cases of mental retardation per 100 rad could be directly applicable to a U.S. population. In this case the risk estimate would be about:

4 cases of mental retardation per rad per 1000 live births.

B. Estimate of Incidence Per Rad Based on the Doubling Dose

The Otake and Schull report (Ot83) suggested the doubling dose for mental retardation was about 2 rad, fetal absorbed dose or about a 50 percent increase in mental retardation per rad. It would seem reasonable that this doubling dose would apply only to idiopathic cases of mental retardation caused during gestation. That is those which have no known genetic, viral, bacterial, etc. cause.

Data from studies of the prevalence of mental retardation in school age populations in developed countries suggest a prevalence of 2.8 cases/1000 (Uppsala County, Sweden) to 7.4 cases/1000 (Amsterdam, Holland) of severe mental retardation, with a mean of about 4.3 ± 1.3 cases/1000 (Sta84). Where data is available for males and females separately, the male rate is about 30 percent higher than the female rate (Sta84). Historically, the prevalence of mild mental retardation has been 6 to 10 times greater than that of severe mental retardation. But, in recent Swedish studies, the rates of prevalence of mild and severe mental retardation have been similar (Sta84). This was suggested to be due to a decline in the "cultural-familial syndrome". That is, improved nutrition, decline in infection and diseases of childhood, increased social and intellectual stimulation, etc., combined to reduce the proportion of nonorganic mental retardation and, therefore, the prevalence of mild mental retardation (Sta84).

In studies of the causes of mental retardation, 23 percent to 42 percent of the mental retardation has no identified cause (Gu77, Haa81, St84). It is this portion of the mental retardation which may be susceptible to increase due to radiation exposure of the embryo/fetus. In that case, the prevalence of idiopathic mental retardation would be 0.6 to 3.1 cases per 1000 of severe mental retardation and perhaps an equal number of cases of mild mental retardation.

For purposes of estimating the effects of radiation exposure of the embryo/fetus a risk of spontaneous idiopathic mental retardation of 1 to 6 per 1000 will be used. If this spontaneous idiopathic mental retardation can be increased by radiation the estimate would be:

(1 to 6 cases per 1000 live births)(0.5 increase per rad)

or about 0.5 to 3 cases of mental retardation per rad per 1000 live births.

This estimate may be biased low. This occurs because mental retardation induced during gestation is often associated with high childhood death rate (Sta84). If this is generally true for idiopathic causes of mental retardation, it would cause an underestimation of the risk.

C. Estimate of Incidence Per Rad Based on Incidence of Microcephaly

(1) 2.275 percent of live born children will have a head circumference 2 standard deviations or more smaller than average, 0.621 percent will have a head circumference 2.5 standard deviations or more smaller than average and 0.135 percent will have a head circumference 3 standard deviations or more smaller than average, (statistical estimate based on a normal distribution).

(2) There is evidence in a nonselected group of 9,379 children that mental retardation can be estimated using the incidence of microcephaly, even though head circumference in the absence of other supporting data, e.g. height or proportion, is an uncertain indicator of mental retardation. Based on a study of 9,379 children, Nelson and Deutschberger (Neb70) concluded that about half of the children with a head circumference 2.5 standard deviations or more smaller than average had IQs of 79 or lower. Since 0.67 percent of those studied were in this group, the observed number is about what would be expected based on the normal distribution of head size in a population, 0.62 percent. The estimated incidence of mental retardation per live birth in a population would be:

$$(6.7 \text{ cases of microcephaly per 1000 live births}) \times$$

$$\left(0.5 \frac{\text{cases of mental retardation}}{\text{case of microcephaly}}\right)$$

or about 3.4 cases of mental retardation per 1000 live births.

(3) A first approximation of risk of mental retardation might then be:

$$(3.4 \text{ cases of mental retardation per 1000 live births}) \times$$

$$(0.5 \text{ increase per rad})$$

or about 2 cases of mental retardation per 1000 live births per rad.

Both microcephaly and mental retardation were increased in Japanese survivors (Woa65, Wob66). About half of those with head sizes 2 or more standard deviations smaller than average had mental retardation (RERF78), a result similar to that observed by Nelson and Deutschberger (Neb70). Therefore, the estimate above based on the incidence of microcephaly in a population should be a reasonable estimate of the risk due to radiation.

Summary of the Calculated Risk of Mental Retardation

The risk of increased mental retardation per rad of embryo/fetus exposure during the 8 to15 week gestational period estimated above ranges from about 5×10^{-4} to 4×10^{-3} cases per live birth, the largest being a direct estimate. The geometric mean of these estimates is 1.4×10^{-3}, the arithmetic mean is 2.4×10^{-3} cases per live birth.

All the estimates derived above by any of the three methods are in the same range as an earlier UNSCEAR (UNSCEAR77) estimate of an increase of 1×10^{-3} cases of mental retardation per rad per live birth. The UNSCEAR estimate, however, did not consider gestational age at the time of exposure. The Otake and Schull report (Ot83) did address gestational age and estimated a higher risk, but a narrower window of susceptibility.

If the estimates are applicable, the 15 mrad of low-LET background radiation delivered during the 8 to 15 week gestational age sensitive period could induce a risk of 6×10^{-5} to 7.5×10^{-6} cases of mental retardation per live birth. This can be compared to an estimate of a spontaneous occurrence of 1.5×10^{-2} to 3.4×10^{-3} cases of mental retardation per live birth.

Japanese A-bomb survivors exposed in utero also showed a number of structural abnormalities and, particularly in those who were microcephalic, retarded growth (Woa65). No estimate has been made of the radiation-related incidence or dose-response relationships for these abnormalities, because of the small number of cases. UNSCEAR (UNSCEAR77) made a very tentative estimate based on animal studies that the increased incidence of recognizable structural abnormalities in animals may be 5×10^{-3} cases per R per live born, but stated that projections to humans was unwarranted. In any event, the available human data cannot show if the risk estimates derived from high dose animal data overestimates the risk in humans.

It should be noted that all of the above estimates are based on high dose rate low-LET exposure. UNSCEAR in 1977 also investigated the dose-rate question and stated:

> "In conclusion, the majority of the data available for most species indicate a decrease of the cellular and malformative effects by lowering the dose rate or by fractionating the dose. However, deviations from this trend have been well documented in a few instances and are not inconsistent with

> the knowledge about mechanisms of the teratogenic effects. It is therefore impossible to assume that dose rate and fractionation factor have the same influence on all teratological effects." (UNSCEAR77).

From this analysis, EPA has concluded that the range of risk is 4×10^{-3} to 5×10^{-4} cases of mental retardation per live birth per rad of low-LET radiation delivered between weeks 8 and 15 of gestation, with no threshold identified at this time.

At this time, no attempt can be made to estimate total teratogenic effects. However, it should be noted that the 1977 UNSCEAR estimate from animals was 5×10^{-3} cases of structural abnormalities per R per live birth (about the same number per rad of low-LET). This estimate must be viewed as a minimum one since it is based, to a large extent, on observation of grossly visible malformations. Differences in criteria for identifying malformations have compounded the problem, and questions of threshold and species differences have made risk projection to humans unwarranted.

8.6.7 Nonstochastic Effects

Nonstochastic effects, those effects that increase in severity with increasing dose and may have a threshold, have been reviewed in the 1982 UNSCEAR report (UNSCEAR82). In general, acute doses of 10 rad low-LET radiation and higher are required to induce these effects. It is possible that some of the observed effects of in utero exposure are nonstochastic, e.g., the risk of embryonic loss, estimated to be 10^{-2} per R (UNSCEAR77) following radiation exposure soon after fertilization. However, there are not enough data to address the question. Usually, no nonstochastic effects of radiation are expected at environmental levels of radiation exposure.

8.7 Radiation Risk - A Perspective

To provide a perspective on the risk of fatal radiogenic cancers and the hereditary damage due to radiation, we have calculated the risk from background radiation to the U.S. population using the risk coefficients presented in this chapter and the computer codes described in Addendum B. The risk resulting from background radiation is a useful perspective for the risks caused by emissions of radionuclides. Unlike cigarette smoking, auto accidents, and other measures of common risks, the risks resulting from background radiation are neither voluntary nor the result of alcohol abuse. The risk caused by background radiation is very largely unavoidable; therefore, it is a good benchmark for judging the estimated risks from radionuclide emissions. Moreover, to the degree that the estimated risk of radionuclides is biased, the same bias is present in the risk estimates for background radiation.

Low-LET background radiation has three major components: cosmic radiation, which averages to about 28 mrad per year in the U.S.; terrestrial sources, such as radium in soil, which contributes an average of 26 mrad per year (NCRP75); and the low-LET dose resulting from internal emitters. The latter differs between organs, to some extent, but for soft tissues is about 24 mrad per year (NCRP75). Fallout from nuclear weapons tests, naturally occurring radioactive materials in buildings, etc., contribute something like another 10 mrem for a total low-LET whole-body dose of about 90 mrad per year. The lung and bone receive somewhat larger doses resulting from high-LET radiations; see below. Although extremes do occur, the distribution of this background annual dose to the U.S. population is relatively narrow. A population weighted analysis indicates that 80 percent of the U.S. population would receive annual doses that are between 75 mrad per year and 115 mrad per year (EPA81).

As outlined in Section 8.2, the BEIR-3 linear models yield, for life time exposure to low-LET radiation, an average life time risk of fatal radiogenic cancer of 280 per 10^6 person rad. Note that this average is for a group having the age and sex specific mortality rates of the 1970 U.S. population. We can use this datum to calculate the average life time risk due to low-LET background radiation as follows. The average duration of exposure in this group is 70.7 years and at 9×10^{-2} rad per year, the average life time dose is 6.36 rad. The risk of fatal cancer per person in this group is:

$$\frac{280 \text{ fatalities}}{10^6 \text{ person rad}} \times 6.36 \text{ rem} = 1.78 \times 10^{-3}$$

or about 0.18 percent of all deaths. The vital statistics we use in our radiation risk analyses indicate that the probability of dying due to cancer in the U.S. due to all causes is about 0.16, i.e. 16 percent. Thus the 0.18 percent result for the BEIR-3 linear dose response model indicates that about 1 percent of all U.S. cancer is due to low-LET background radiation. The BEIR-3 linear quadratic model indicates that about 0.07 percent of all deaths are due to low-LET background radiation or about 0.4 percent of all cancer deaths.

The information in Volume 2 of this BID indicates that airborne radioactive emissions may cause additional cancer risks comparable to those risks due to background radiation. For example, the models described in Chapters 6 and 7 indicate that emission from the Monsanto Plant in Idaho could result in lung doses to nearby individuals of about 30 mrad per year due to inhaled alpha particle emitters. A 30 mrad annual dose of alpha radiation results in a dose equivalent rate to the lung of 600 mrem per year.*

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*The dose equivalent rate to other organs is 30-100 times smaller.

Table 8.3-1 indicates a risk of 460 fatalities per 10^6 organ rad for alpha emitters in lung tissue. The life time cancer from this exposure is:

$$\frac{460 \text{ fatalities}}{10^6 \text{ organ rad}} \times \frac{0.03 \text{ rad}}{\text{y}} \times 70.7\text{y} = 0.98 \times 10^{-3}$$

c.f. Table 6.3-13 in Volume 2 of this BID. This is twice the risk due to low-LET background radiation calculated by means of the BEIR-3 linear quadratic model and more than half of the risk calculated by means of the BEIR-3 linear model.

The 1982 UNSCEAR report indicates that the average annual dose to the endosteal surfaces of bone due to naturally occurring high-LET alpha radiation is about 6 mrad per year or, for a quality factor 20, 120 mrem per year (UNSCEAR82). Table 8.3-1 indicates that the life time risk of fatal bone cancer due to this portion of the naturally occurring radiation background is

$$\frac{20 \text{ cases}}{10^6 \text{ person rad}} \times \frac{0.006 \text{ rad}}{\text{year}} \times 70.7 \text{ years} = 8.5 \times 10^{-6}$$

The exposure due to naturally occurring background radon-222 progeny in the indoor environment is not well known. The 1982 UNSCEAR report lists for the U.S. an indoor concentration of about 0.004 working levels (15 Bq m^{-3}, (UNSCEAR82). This estimate is not based on a national survey and is known to be exceeded by as much as a factor of ten or more in some houses. However, as pointed out in UNSCEAR82, the national collective exposure is not too dependent on exceptions to the mean concentration.

Assuming 0.004 WL is a reasonable estimate for indoor exposure to radon-222 progeny, the EPA exposure model outlined in 8.4 yields a mean life time exposure, indoors, of 6.7 WLM. In Section 8.24 two risk coefficients for lung cancer due to radon progeny are presented. The largest, 700 fatalities per 10^6 person WLM, yields a probability of death of 0.0047. That is, about one-half percent of all deaths are estimated as due to naturally occurring indoor radon progeny. We note that this is comparable to the 1 percent fatality incidence estimated above for low-LET background radiation. The smaller risk coefficient listed in 8.4, 300 fatalities per 10^6 person WLM, implicates radon progeny in about 0.2 percent of all deaths. The reader is cautioned, however, that these risk estimates only apply to the U.S. population taken as a whole, i.e. men and women, smokers and nonsmokers. While we believe they are reasonable estimates for the U.S. 1970 population in which the vast majority of the lung cancer mortality occurred in male smokers, we do not believe these risk estimates can be applied indiscriminately to women or nonsmokers. As noted in Section 8.4, the risk to these groups may not be comparable.

The spontaneous incidence of serious congenital and genetic abnormalities has been estimated to be about 105,000 per 10^6 live births, about 10.5 percent of live births (NAS80, UNSCEAR82). The low-LET background radiation dose of about 90 mrad/year in soft tissue results in a genetically significant dose of 2.7 rad during the 30 year reproductive generation. Since this dose would have occurred in a large number of generations, the genetic effects of the radiation exposure are thought to be an equilibrium level of expression. As noted in 8-6, since genetic risk estimates vary by a factor of 20 or more EPA uses a log mean of this range to obtain an average value for estimating genetic risk. Based on this average value, the background radiation causes 700 to 1000 genetic effects per 10^6 live births, depending on whether or not the oocyte is as sensitive to radiation as the spermatogonia, see 8.6. This result indicates that about 0.67 to 0.95 percent of the current spontaneous incidence of serious congenital and genetic abnormalities may be due to the low-LET background radiation.

The gonadal dose and genetic risk from airborne radionuclide emissions is usually quite small. For example, the 30 year gonodal dose due to the Monsanto plant, referred to above, is about 0.8 mrad, high LET, and 0.3 mrad, low LET. From Table 8.6-5, the risk of serious hereditary disorder from these doses, assuming equal male and female sensitivity is:

$$\frac{7400}{10^6 \text{ live births}} \times 0.8 \times 10^{-3} = 5.9 \times 10^{-6} \text{ high LET}$$

$$\frac{370}{10^6 \text{ live births}} \times 0.3 \times 10^{-3} = 0.1 \times 10^{-6} \text{ low LET}$$

or about 6 cases in a million live births. This is the total for all generations. Ten to twenty percent of these might occur in the first generation after exposure of the parents. The total for all generations is a hundred times smaller than the estimated cancer risk from this source, a result that is quite general for radionuclide air emissions of particulates.

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Chapter 9: SUMMARY OF DOSE AND RISK ESTIMATES

9.1 Introduction

This chapter summarizes the calculated doses and risks for the facilities analyzed in Chapters 2 through 7 of Volume II. Also, overall uncertainties in these estimates are discussed.

Four separate steps are involved in estimating the health impact of a specific source of radioactivity: (1) measurement of emissions of radionuclides to air from the source, (2) estimation of the radionuclide concentration and annual intake of radionuclides at various locations, (3) calculation of the estimated dose and risk resulting from a unit intake or unit concentration of radioactivity in the environment, and (4) a means of scaling the risk estimates to match the specific source. In EPA's analysis, each step is associated with a computer code that performs the necessary calculations; the relationship of these codes is illustrated in Figure A-1 (Addendum A).

EPA uses the AIRDOS-EPA code (Mo79, Ba81) to analyze radionuclide emissions into air from a specific source. The results of this analysis are estimates of air and ground surface radionclide concentrations, intake rates via inhalation of air, and ingestion of radioactivity via meat, milk, and fresh vegetables. Chapter 6 presents a description of the techniques used and their limitations. The atmospheric and terrestrial transport models used in the code, their implementation, and the applicability of the code to different types of emissions are described in detail in Mo79.

The computer code used to calculate dose and risk is RADRISK (Dub84, Su81, Dua80). RADRISK calculates the radiation dose and risk resulting from an annual unit, e.g., 1 pCi/y, intake of a given radionuclide or the risk resulting from external exposure to a unit, e.g., 1 pCi/m^3, 1 pCi/m^2, concentration of radionuclide in air or on ground surface. Since both dose and risk models are linear, the unit dose and risk results can then be scaled to reflect the conditions associated with a specific source. The assessment of radiation doses is discussed in Chapter 7; Chapter 8 discusses estimating the risk of health effects.

Once the radionuclide intakes and concentrations are calculated for a specific source by means of the environmental transport code, it is necessary to scale the dose and risk values resulting from a unit intake or concentration to the intake and concentration values predicted by the transport code. As shown in Figure A-1 (Addendum A), the DARTAB computer code (Be81) performs this step using RADRISK unit doses and risks and AIRDOS-EPA concentrations and intakes. DARTAB is independent of both the environmental transport code, e.g., AIRDOS-EPA, and the dosimetric and health effects code, e.g., RADRISK. This eliminates redundant dose/risk calculations and the need for extraneous coding to calculate doses and health impacts in each environmental transport code.

9.2 Doses and Risks for Specific Facilities

Tables 9.2-1 and 9.2-2 are summaries of the doses and risks to critical groups of individuals and populations in the vicinity of facilities that discharge radioactive emissions. Data for selected facilities from each category are presented in the order they are presented in Chapters 2 through 7 of Volume II.

These dose and risk values were estimated using the environmental transport codes of AIRDOS-EPA, the dose and risk tables of DARTAB and the risk estimates that compose the RADRISK code. More detailed information, including a description of the facility, the processes causing the emissions, estimates of rates of emission, and estimates of doses and risks that result to individuals and populations are found in the respective chapters of Volume II.

9.3 Overall Uncertainties

Although the doses and risks presented in Tables 9.2-1 and 9.2-2 seem well defined and sometimes given to more than one significant figure, there are considerable uncertainties that persist when trying to fix their exact value. The individual uncertainties in the components which lead to the results in Tables 9.2-1 and 9.2-2 have been previously discussed. Source term measurement errors were discussed in Chapter 4; possible errors introduced in evaluating movement from the source through various pathways were discussed in Chapter 6; variations which could be introduced in the calculation of doses and dose rates were evaluated in Chapter 7; finally, Chapter 8 discussed the potential errors that could be introduced in the risk calculations.

9.3.1 Emission and Pathway Uncertainties

Measurement of emissions from sources have been estimated in Chapter 4 to be valid within a factor of 1.4.

In the evaluation of pathways, the uncertainties in results predicted by the atmospheric dispersion models make the most significant contribution. As discussed in Section 6.2.3, the studies by Little (Li79) and Miller (Mi82) indicate that for average annual concentrations, an uncertainty of approximately a factor of 2 for locations within 10 km of the release could be expected. Inasmuch as nearby locations to releases are of greatest concern, this uncertainty value is the most appropriate.

Table 9.2-1. Doses and risks to nearby individuals

Facility	Tissue(a)	Dose rate (mrem/year)	Lifetime(b,c,d) Risk (deaths/10^{-6} persons)
DOE Facilities			
Feed Materials Production Center	lung	88	100 (100)
Oak Ridge Reservation	lung	50	100 (100)
Portsmouth Gaseous Diffusion Plant	bone surface	11	20 (20)
Savannah River Plant	thyroid	4.9	40 (20)
NRC Facilities			
Research and test reactor(e)	average all organs	1	20 (8)
Accelerator(f)	average all organs	0.0001	0.002 (0.0008)
Radiopharmaceutical suppliers(e)	thyroid	0.3	0.2 (0.1)
AFRRI(g)	average all organs	0.005	0.09 (0.04)
U.S. Army facility	spleen	0.03	0.4 (0.2)
U.S. Navy facility(e)	average all organs	0.02	0.3 (0.1)
Radiation source manufacturer(e)	average all organs	0.2	4 (2)
Coal Fired Boilers(e)			
Utility boilers (rural)	bone surface	5	30 (10)
Industrial boilers	bone surface	0.4	0.6 (0.5)
Uranium Mine(e)			
Ground level release (at 2000 meters)	lung	No dose available	10,000 (5,000)(h)
Plume rise release (at 2000 meters)	lung	No dose available	1,000 (500)(h)
Phosphate Industry			
Drying and grinding(e)	bone surface	15	10
Wet process fertilizer	bone surface	2	2
Elemental phosphorus			
Pocatello, Idaho	lung	290	500
Soda Springs, Idaho	lung	610	1000
Mineral Extraction Industry(e)			
Aluminum reduction plant	kidney	1.2	0.8
Copper smelter	lung	0.2	0.3
Zinc smelter	bone surface	0.02	0.02
Lead smelter	lung	4.8	8

(a) Organ with highest annual dose.

(b) Risk is that due to the total exposure not just that due to highest organ. This value represents the excess cancers in a lifetime for organ dose rates shown at offsite points of highest risk.

(c) The risk estimates in parentheses include a dose rate reduction factor of 2.5 for low-LET radiation, as described in Chapter 8 (Volume I) of this report.

(d) Risks are expressed per million population; for individual risks multiply each value by 10^{-6}.

(e) Reference facility.

(f) 6 mev Van de Graaff.

(g) Armed Forces Radiobiology Research Institute.

(h) The values in the first column are based on BEIR-3 (NAS80), NRPB (NRPB82), and EPA models (Co78, El84, Mo79); the values in parentheses are based on UNSCEAR (UNSCEAR82) and ICRP (Of80) risk estimates (see Chapter 8, Volume I).

Table 9.2-2. Doses and risks to regional population

Facility	Organ[a]	Collective dose rate (pers-rem/year)	Risk[b,c] (fatal cancers/year)
DOE Facilities			
Feed Materials Production Center	lung	440	0.01 (0.01)
Oak Ridge Reservation	lung	212	0.008 (0.006)
Portsmouth Gaseous Diffusion Plant	bone surface	35	<0.001
Savannah River Plant	thyroid	120	0.03 (0.01)
NRC Facilities			
Research and test reactor	average all organs	340	0.1 (0.04)
Accelerator[d][e]	average all organs	0.0006	<0.001
Radiopharmaceutical suppliers[d]	thyroid	3	<0.001
AFRRI[f]	average all organs	0.002	<0.001
U.S. Army facility	spleen	0.09	<0.001
U.S. Navy facility[d]	average all organs	0.09	<0.001
Radiation source manufacturer[d]	average all organs	8	0.002 (<0.001)
Coal Fired Boilers[d]			
Utility boilers (rural)	bone surface	140	0.005 (0.003)
Industrial boilers	bone surface	90	0.003
Uranium Mine[d]	lung	No dose available	0.06 (0.02)[g]
Phosphate Industry			
Drying and grinding[d]	bone surface	110	0.001
Wet process fertilizer	bone surface	41	0.0005
Elemental phosphorus			
Pocatello, Idaho	lung	1170	0.03
Soda Springs, Idaho	lung	750	0.02
Mineral Extraction Industry[d]			
Aluminum reduction plant	kidney	4.1	<0.001
Copper smelter	lung	0.95	<0.001
Zinc smelter	bone surface	2.5	<0.001
Lead smelter	lung	69	0.002

---

[a] Organs with highest annual dose.

[b] Fatal cancers in regional population per year of operation of facility for population exposure rate shown.

[c] The risk estimates in parentheses include a dose rate reduction factor of 2.5 for low-LET radiation, as described in Chapter 8 (Volume I) of this report.

[d] Reference facility

[e] 6 mev Van de Graaff.

[f] Armed Forces Radiobiology Research Institute.

[g] The values in the first column are based on BEIR-3 (NAS80), NRPB (NRPB82), and EPA models (Co78, El84, Mo79); the values in parentheses are based on UNSCEAR (UNSCEAR82) and ICRP (Of80) risk estimates (see Chapter 8, Volume I).

9.3.2 Dose Uncertainties

As discussed in Chapter 7 and summarized in Section 7.7, dose uncertainties are much less than would be implied by sensitivity analyses of maximum ranges of variables. The large dose ranges possible because of variation in individual characteristics must be modified by consideration of the narrower ranges indicated by studies of real populations; the dose range resulting from age dependence appears to be small for lifetime exposures, and the range resulting from experimental error is negligible by comparison. Based on these observations, it is reasonable to estimate that EPA's doses calculated on the basis of unit intakes or unit concentrations should be accurate within a factor of three or four. Much of the "uncertainty" in the dose calculation is not caused by parameter error but reflects real differences in individual characteristics within the general population. Therefore, the uncertainty in the dose estimates cannot be dissociated from specification of the segment of the population to be protected.

9.3.3 Risk Uncertainties

The uncertainties in estimating risk have been discussed in Chapter 8. Table 8.5-1 ranks and estimates the degree of uncertainty introduced by various sources in estimating the risk of cancer. The uncertainties listed in Table 8.5-1 are largely independent of each other and therefore unlikely to be correlated in sign. Their root mean square sum is about 300 percent, indicating the expectation that calculated risks would be within a factor of three or so of the true value. (This result is likely to be somewhat low because it does not include consideration of the uncertainty introduced by the bias in the A-bomb dosimetry or by the constrained regression analysis used by the BEIR-3 Committee.)

9.3.4 Overall Uncertainty

As indicated in the previous discussion, the individual uncertainties which combine to provide a basis for the overall uncertainty in risk evaluation are the following:

- Emission estimates are valid within a factor of 1.4*
- Air concentration estimates are valid within a factor of 2
- Dose calculations should be valid within a factor of 3 or 4
- Risk calculations should be valid within a factor of 3.

If these uncertainty estimates are independent and uncorrelated and can reasonably be considered to estimate the 2σ fractile of a log normal distribution, then the overall uncertainty in EPA's risk estimation can be estimated as a factor of about 7*. That is the maximum expected variation would range from about 15 percent to 700 percent of the nominal value.

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*If the nominal value is multiplied or divided by the factor to give a range, the true value is expected to be within that range.

The various uncertainties, however, may not be uncorrelated or independent. In this case, the overall uncertainty is likely to be less than predicted by the above procedure.

EPA concludes that risk estimates in this Background Information Document are accurate within a factor of 10. This estimate of uncertainty is believed representative of state-of-the-art procedures for estimating risks due to airborne radionuclide emissions.

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*$\exp\{[\ln^2(1.4) + \ln^2(2) + \ln^2(4) + \ln^2(3)]^{1/2}\}$.

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ADDENDUM A

COMPUTER CODES USED BY EPA TO ASSESS DOSES FROM RADIATION EXPOSURE

ADDENDUM A: COMPUTER CODES USED BY EPA TO ASSESS DOSES FROM RADIATION EXPOSURE

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ADDENDUM A: COMPUTER CODES USED BY EPA TO ASSESS DOSES FROM RADIATION EXPOSURE

A.1 Introduction

This addendum (to Chapter 7) provides a brief overview of the computer codes used by the Environmental Protection Agency (EPA) to assess the health risk from radiation exposures. It describes how the basic dose calculations are performed. Comprehensive descriptions of the various parts of this methodology have been published in a series of reports by the Oak Ridge National Laboratory and the Environmental Protection Agency (Dub84, Be81, Ba81, Moa79). The risk estimates in current use are described in Chapter 8 and reflect the change from the BEIR-1 report (NAS72) to the BEIR-3 report (NAS80).

Three separate steps are required to estimate the health impact of a specific source of radioactivity: (1) estimate at various locations the radionuclide concentration and annual intake of radionuclides resulting from specific sources of radioactivity in the environment, (2) calculate the estimated dose and risk resulting from a unit intake or unit concentration of radioactivity in the environment, and (3) use a means of scaling the risk estimates to match the specific source. In EPA's analysis, each step is associated with a computer code that performs the necessary calculations, as illustrated in Figure A-1.

A.2 Overview of the EPA Analysis

The computer code used to calculate dose and risk is RADRISK (Dub84, Su81, Dua80). RADRISK calculates the radiation dose and risk resulting from an annual unit intake of a given radionuclide or the risk resulting from external exposure to a unit concentration of radionuclide in air or on ground surface. Since both dose and risk models are linear, the unit dose and risk results can then be scaled to reflect the exposure associated with a specific source.

As outlined in Chapter 7, estimates of the annual dose rate to organs and tissues of interest are calculated using, primarily, models recommended by the International Commission on Radiological Protection (ICRP79, ICRP80). Because EPA usually considers lifetime exposures to a general population, these dose rates are used in conjunction with a life table analysis of the increased risk of cancer resulting from radiation (Co78). This analysis, described in Addendum B, takes account of both competing risks and the age of the population at risk.

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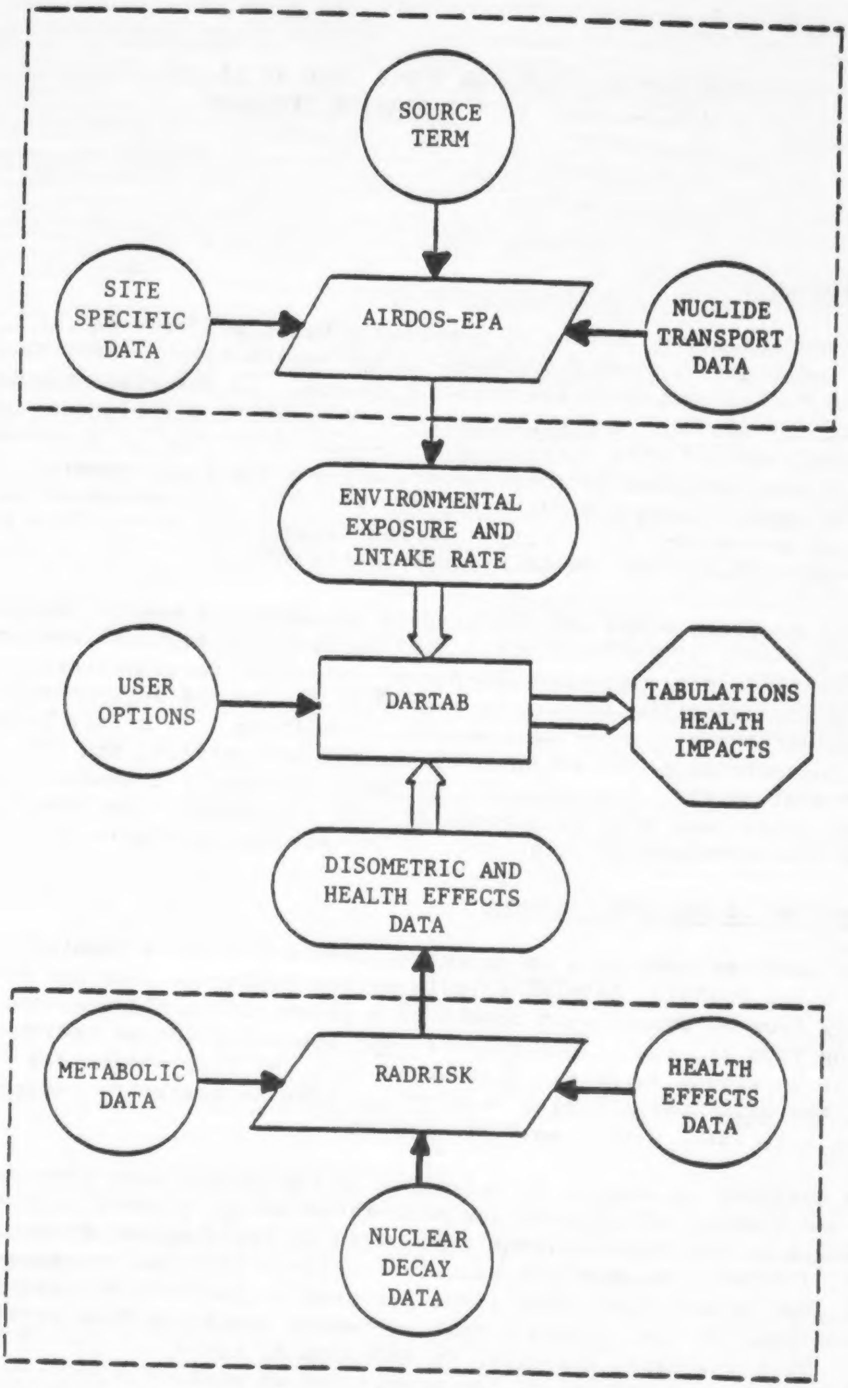
SOURCE TERM
SITE SPECIFIC DATA
AIRDOS-EPA
NUCLIDE TRANSPORT DATA
ENVIRONMENTAL EXPOSURE AND INTAKE RATE
USER OPTIONS
DARTAB
TABULATIONS HEALTH IMPACTS
DISOMETRIC AND HEALTH EFFECTS DATA
METABOLIC DATA
RADRISK
HEALTH EFFECTS DATA
NUCLEAR DECAY DATA

Figure A-1. Assessment of radiological health impacts.

Various computer codes are available to predict how radionuclides are transported through environmental pathways. As noted in Figure A-1, EPA uses the AIRDOS-EPA code (Moa79, Ba81) to analyze the transport of radionuclide emissions into air from a specific source. The results of this analysis are estimates at various distances from the source of air and ground surface radionuclide concentrations, intake rates via inhalation of air, and ingestion of radioactivity via meat, milk, and fresh vegetables. The atmospheric and terrestrial transport models used in the code, their implementation, and the applicability of the code to different types of emissions are described in Chapter 6.

A.3 Dose Rates from Internal Exposures

Internal exposures occur when radioactive material is inhaled or ingested. The RADRISK code implements contemporary dosimetric models to estimate the dose rates at various times to specified reference organs in the body from inhaled or ingested radionuclides. The dosimetric methods in RADRISK are adapted from those of the INREM II code (Ki78), based primarily on models recommended by the International Commission on Radiological Protection (ICRP79). The principal qualitative difference is that RADRISK computes dose rates to specified organs separately for high and low linear energy transfer (LET) radiations, whereas INREM II calculates the committed dose equivalent to specified organs. The time-dependent dose rates are used in the life table calculations of RADRISK.

In RADRISK, the direct intake of each nuclide is treated as a separate case. For chains, the ingrowth and dynamics of daughters in the body after intake of a parent radionuclide are considered explicitly in the calculation of dose rate. Consideration is also taken of different metabolic properties of the various radionuclides in a decay chain.

The dose rate $\dot{D}_i(X,t)$ to target organ X at time t due to radionuclide i ($1 \le i \le N$) residing in organs $Y_1, Y_2, \ldots, Y_m$ is a measure of the energy deposited annually in a given mass of tissue as a result of radioactive decay, and is computed as:

$$\dot{D}_i(X;t) = \sum_{k=1}^{m} \dot{D}_i(X \leftarrow Y_k;t) \qquad \text{(A-1)}$$

where

$$\dot{D}_i(X \leftarrow Y_k;t) = S_i(X \leftarrow Y_k)A_{ik}(t) \quad , \qquad \text{(A-2)}$$

$A_{ik}(t)$ = activity of radionuclide i in organ Y_k at time t measured from the initial intake of i into the body,

$S_i(X \leftarrow Y_k)$ = average dose rate to target organ X per unit activity of the radionuclide i uniformly distributed in source organ Y_k (Sn74, Dua80).

The summation is taken over all source organs Y. Implicit in the definitions is the assumption of uniform distribution of activity of radionuclide i in each source organ, as is the assumption of averaging the dose rate over the mass of the target organ. Although estimates of dose to an organ include contributions from activity distributed throughout the body (for penetrating radiations), activity within that organ generally contributes the principal component of dose [i.e., $\dot{D}_i(X \leftarrow X;t)$ is the principal component of $\dot{D}_i(X;t)$].

The time rate of change of activity in the body is modeled by a system of ordinary differential equations, with each equation describing the rate of change of activity in a conceptual compartment of the body. For radionuclides that are part of a decay series there may be formation of radioactive daughters in a given compartment that have different chemical and physical properties from those of the parent. Unlike the models given in ICRP80, the specific metabolic properties of the daughter are evaluated when they differ from those of the parent. This refinement is under active consideration by ICRP experts. In almost all cases, doses to soft tissues calculated on this basis differ only slightly, if at all, from ICRP80 dose estimates, but the difference is large for some radionuclides when the parent is incorporated into bone, for example lead-210. For this radionuclide the ICRP80 model has been used without any modifications.

The pathways in the body by which activity is assumed to move were illustrated in Chapter 7. Except for radon daughters, which are considered separately, inhaled activity is assumed to be originally deposited in the lungs (distributed among the nasal-pharyngeal, tracheobronchial, and pulmonary regions), whereas ingested activity is originally deposited in the stomach. From the lungs, activity may be absorbed by the bloodstream or migrate to the stomach. Activity in the stomach may proceed through the small intestine, upper large intestine, and lower large intestine; activity may be absorbed by the bloodstream from any of these four segments, although only absorption from the small intestine is considered in this study.

The activity, $A_{ik}(t)$, of nuclide i in organ k may be divided among several "pools" or "compartments", denoted here by the subscript ℓ. Each differential equation describing the rate of change of activity within a compartment is a special case of the equation:

$$\dot{A}_{i\ell k} = -(\lambda_i^R + \lambda_{i\ell k}^B)A_{i\ell k} + c_{i\ell k}\left(\lambda_i^R \sum_{j=1}^{i-1} B_{ij} \sum_{r=1}^{L_{jk}} A_{jr} + P_{ik}\right), \quad \ell=1,\ldots,\ell_{ik} \qquad \text{(A-3)}$$

where

$\dot{A}_{i\ell k}$ = activity of radionuclide i in compartment ℓ of organ k,

$L_{i\ell k}$ = number of exponential terms in the retention function for nuclide i in organ k,

B_{ij} = branching ratio of nuclide j to nuclide i,

λ_i^R = rate coefficient (time^{-1}) for radiological decay of nuclide i,

$\lambda_{i\ell k}^B$ = rate coefficient (time^{-1}) for biological removal of nuclide i from compartment ℓ of organ k,

$c_{i\ell k}$ = fractional coefficient for nuclide i in the ℓ-th compartment of organ k,

P_{ik} = inflow rate of nuclide i into organ k.

If the inflow rate P_{ik} remains constant, the equations may be solved explicitly for $A_{ik}(t)$ as described by Killough, Dunning, and Pleasant (Ki78). In many cases the inflow into a compartment will not be a constant rate over a long period of time. To handle this problem, the time interval over which solution of the activity equation is desired (e.g., 110 years) is divided into 1-year subintervals. The inflow rate on each subinterval is then taken to be that constant value which would yield the total activity flowing out of the preceding compartment(s) during the same subinterval.

The model used in RADRISK for particulate deposition and retention in the respiratory tract is the ICRP task group lung model (Mob66, ICRP72). In this model, shown in Chapter 7, there are four major regions: the naso-pharyngeal, tracheobronchial, pulmonary, and lymphatic tissues. A fraction of the inhaled activity is initially deposited in each of the naso-pharyngeal, tracheobronchial, and pulmonary regions. The material clears from the lung to the blood and the gastrointestinal tract, also as shown in Chapter 7. Deposition and clearance of inspired particulates in the lung are controlled by the particle size and solubility classification.

The size distribution of the particles is specified by the activity median aerodynamic diameter (AMAD); in this document, all particulates are assumed to have an AMAD equal to 1.0 micron unless otherwise stated. The model employs three solubility classes, based on the chemical properties of the nuclide; classes D, W, and Y correspond to rapid (days), intermediate (weeks), and slow (years) clearance, respectively, of material deposited in the respiratory passages. Inhaled nonreactive, i.e., noble, gases are handled as a special case.

Movement of activity through the gastrointestinal (GI) tract is simulated with a catenary model, consisting of four segments: stomach, small intestine, upper large intestine, and lower large intestine. Exponential outflow of activity from each segment into the next or out of the system is assumed. Outflow rate constants are calculated from the transit times of Eve (Ev66). Although absorption may occur from any combination of the four segments, only activity absorbed into the blood from the small intestine is normally considered; the fractional absorption from the small intestine into the blood is traditionally denoted f_1.

Activity absorbed by the blood from the GI or respiratory tract is assumed to be distributed immediately to systemic organs. The distribution of activity to these organs is specified by fractional uptake coefficients. The list of organs in which activity is explicitly distributed (herein termed source organs) is element-dependent, and may include such organs as bone or liver where sufficient metabolic data are available. This list is complemented by an additional source region denoted as OTHER, which accounts for that systemic activity not distributed among the explicit source organs; uniform distribution of this remaining activity within OTHER is assumed.

Radioactive material that enters an organ may be removed by both radioactive decay and biological removal processes. For each source organ, the fraction of the initial activity remaining at any time after intake is described by a retention function consisting of one or more exponentially decaying terms.

The metabolic models and parameters employed in the present study have been described by Sullivan et al. (Su81). In most cases, the models are similar or identical to those recently recommended by the ICRP (ICRP79, ICRP80, ICRP81). However, some of this work was performed prior to the publication of these documents, so that differences in model parameters do exist for some radionuclides (Su81). In particular, parameter values that are thought to be more representative of metabolism following low-level environmental exposures, rather than occupational exposures, have been used in this analysis [e.g., f_1=0.2 for uranium in the environment (ICRP79, NAS83)]. For transuranic isotopes, metabolic parameters from the Proposed Guidance on Dose Limits for Persons Exposed to Transuranium Elements in the General Environment (EPA77), related comments (EPA78), and from the National Radiological Protection Board (NRPB82), have been used rather than those from ICRP80. These parameters are listed in Table A-1.

The EPA values were recommended by U.S. experts on transuranic element metabolism at Battelle Pacific Northwest Laboratory (EPA78). The recently adopted National Radiation Protection Board f_1 values for transuranics in the general environment are closer to those values proposed by EPA in 1977 than those currently advocated by ICRP for occupational exposures. Use of these larger f_1 values increases the estimated dose and risk from ingestion of transuranic materials but has little effect on doses following inhalation.

Table A-1. Small intestine to blood transfer fractions, f_1, for transuranic elements

	EPA		NRPB		
	Child	Adult		Child	
Element Isotope	0-12 mo	>12 mo	Adult	0-12 mo	0-3 mo
$\mathrm{Pu}^{\substack{238\\241}}$					
Oxide form	10^{-2}	10^{-3}	10^{-5}(b)	5×10^{-4}(b)	10^{-3}(b)
Nonoxide form	10^{-2}	10^{-3}	5×10^{-4}	5×10^{-3}	10^{-2}
Bio. inc.(a)	5×10^{-2}	5×10^{-3}	5×10^{-4}	5×10^{-3}	10^{-2}
$\mathrm{Pu}^{\substack{239\\240}}$					
Oxide form	10^{-3}	10^{-4}	10^{-5}(b)	5×10^{-4}(b)	10^{-3}(b)
Nonoxide form	10^{-2}	10^{-3}	5×10^{-4}	5×10^{-3}	10^{-2}
Bio. inc.	5×10^{-2}	5×10^{-3}	5×10^{-4}	5×10^{-3}	10^{-2}
Am					
Oxide form	10^{-2}	10^{-3}	5×10^{-4}	5×10^{-3}	10^{-2}
Nonoxide form	10^{-2}	10^{-3}	5×10^{-4}	5×10^{-3}	10^{-2}
Bio. inc.	5×10^{-2}	5×10^{-3}	5×10^{-4}	5×10^{-3}	10^{-2}
Cm					
Oxide form	10^{-2}	10^{-3}	5×10^{-4}	5×10^{-3}	10^{-2}
Nonoxide form	10^{-2}	10^{-3}	5×10^{-4}	5×10^{-3}	10^{-2}
Bio. inc.	5×10^{-2}	5×10^{-3}	5×10^{-4}	5×10^{-3}	10^{-2}
Np	-	10^{-3}	10^{-3}	5×10^{-3}	10^{-2}

(a) Biologically incorporated form.
(b) Hydroxide form.
Source: (EPA77, EPA78, NRPB82).

A.4 Dose Rates from External Exposures

As a result of the penetrating nature of photons, radioactivity need not be taken into the body to deliver a dose to body organs. Energy absorbed from photons emitted by radionuclides residing in the air or on the ground surface may also contribute to the overall risk. Indeed, natural background radiation is an example of an important external exposure, ordinarily contributing the largest component of dose to mankind.

Dose rates to organs of an individual immersed in contaminated air or standing on a contaminated ground surface are computed by the DOSFACTER computer code of Kocher (Ko81). These calculations assume that the radionuclide concentration is uniform throughout an infinite volume of air or area of ground surface, and that the exposed individual is standing on the ground surface. Only photons penetrate the body sufficiently to deliver a significant dose to internal organs, and only doses from photon radiation are considered in this analysis. Beta radiation is far less penetrating and delivers a dose only to the body surface; because skin is not a target tissue of concern in this analysis, no consideration of beta contributions to dose is required. Alpha particles have even less penetration ability, and are also excluded from consideration here.

The photon dose rate factor $\dot{D}_i^{\gamma}$ (X) for a given target organ, X, of an individual immersed in a unit concentration of contaminated air at any time may be expressed as:

$$\dot{D}_i^{\gamma}(X) = c\, K_{pm} \frac{1}{\rho_a} \sum_n f_n^{\gamma} E_n^{\gamma} \left[\frac{(\mu/\rho)_t}{(\mu/\rho)_a}\right]_n G^X \qquad \text{(A-4)}$$

where

ρ_a = density of air,

K_{pm} = 0.5 = particle-medium correction factor,

f_n^{γ} = intensity of n^{th} discrete photon (number/disintegration),

E_n^{γ} = energy of n^{th} photon,

μ/ρ = photon mass energy absorption coefficient, with subscripts "t" and "a" denoting tissue and air, respectively for photons of energy E_n,

G^X = ratio of absorbed dose in organ X to absorbed dose at the body surface,

c = unit conversion proportionality constant.

The terms μ/ρ and G^X are functions of photon energy, E^γ_n.

The photon dose rate factor $\dot{D}^\gamma_{iz}$ (X) to organ X of an individual at a distance z above a unit concentration contaminated ground surface may be computed as:

$$\dot{D}^\gamma_{iz}(X) = 0.5\, c\, K_{pm} \sum_n f^\gamma_n E^\gamma_n [(\mu/\rho)_t]_n \left\{ \int_z^\infty 1/r \exp(-\mu_{an} r)\, dr - [C_{an}/(D_{an}-1)] \exp[(D_{an}-1)\mu_{an} z] \right\} G^X \qquad \text{(A-5)}$$

where

K_{pm} = 1.0 = particle-material correction factor,

μ_{an} = mass attenuation coefficient for the n^th^ discrete photon,

z = height of reference position above ground surface (taken to be 1 meter in this study),

c = unit conversion proportionality constant.

The coefficients C_{an} and D_{an} are functions of the photon energy. A detailed discussion of the derivation of these equations as well as an extensive tabulation of dose rate factors for various radionuclides is presented by Kocher (Ko79, Ko81).

In the analysis here, the dose rate factors described by these equations are scaled to achieve a continuous exposure of 1 pCi/cm^3 for air immersion and 1 pCi/cm^2 for ground surface exposure. Risk estimates for these exposure pathways are based on continuous lifetime exposure to these levels.

Once the radionuclide intakes and concentrations are calculated for a specific source by means of the environmental transport code, it is necessary to scale the dose and risk values due to a unit intake or concentration to the intake and concentration values predicted by the transport code. As shown in Figure A-1, the DARTAB computer code (Be81) performs this step using RADRISK unit doses and risks and AIRDOS-EPA concentrations and intakes. DARTAB is independent of both the environmental transport code, e.g., AIRDOS-EPA, and the dosimetric and health effects code, e.g., RADRISK. This eliminates redundant dose/risk calculations and the need for extraneous coding to calculate doses and health impacts in each environmental transport code.

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ADDENDUM B

MECHANICS OF THE LIFE TABLE IMPLEMENTATION OF THE RISK ESTIMATES

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ADDENDUM B: MECHANICS OF THE LIFE TABLE IMPLEMENTATION OF THE RISK ESTIMATES

B.1 Introduction

This addendum describes the mechanics of the life table implementation of the risk estimates derived in Chapter 8. The calculation is performed as an integral part of the RADRISK code, described in Chapter 7, since time dependent organ dose rates are used.

B.2 Life Table Analysis to Estimate the Risk of Excess Cancer

Radiation effects can be classified as stochastic or nonstochastic (NAS80, ICRP77). For stochastic effects, the probability of occurrence of the effect, as opposed to the severity, is a function of dose; induction of cancer, for example, is considered a stochastic effect. Nonstochastic effects are those health effects for which the severity of the effect is a function of dose; examples of nonstochastic effects include cell killing, suppression of cell division, cataracts, and nonmalignant skin damage.

At the low levels of radiation exposure attributed to radionuclides in the environment, the principal health detriment is the induction of cancers (solid tumors and leukemia), and the expression, in later generations, of genetic effects. In order to estimate these effects, instantaneous dose rates for each organ at specified times are sent to a subroutine adaptation of CAIRD (Co78) contained in the RADRISK code. This subroutine uses annual doses derived from the transmitted dose rates to estimate the number of incremental fatalities in the cohort due to radiation-induced cancer in the reference organ. The calculation of incremental fatalities is based on estimated annual incremental risks, computed from annual doses to the organ, together with radiation risk factors such as those given in the 1980 NAS report BEIR-3 (NAS80). Derivation of the risk factors in current use is discussed in Chapter 8.

An important feature of this methodology is the use of actuarial life tables to account for the time dependence of the radiation insult and to allow for competing risks of death in the estimation of risk due to radiation exposure. A life table consists of data describing age-specific mortality rates from all causes of death for a given population. This information is derived from data obtained on actual mortality rates in a real population; mortality data for the U.S. population during the years 1969-1971 (HEW75) are used throughout this study.

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The use of life tables in studies of risk due to low-level radiation exposure is important because of the time delay inherent in radiation risk. After a radiation dose is received, there is a minimum induction period of several years (latency period) before a cancer is clinically observed. Following the latency period, the probability of occurrence of a cancer during a given year is assumed to be constant for a specified period, called a plateau period. The length of both the latency and plateau periods depends upon the type of cancer.

During or after radiation exposure, a potential cancer victim may experience years of life in which he is continually exposed to risk of death from causes other than incremental radiation exposure. Hence, some individuals will be lost from the population due to competing causes of death, and are not potential victims of incremental radiation-induced cancer.

It is assumed that each member of the hypothetical cohort is exposed to a specified activity of a given radionuclide. In this analysis each member of the cohort annually inhales or ingests 1 pCi of the nuclide, or is exposed to a constant external concentration of 1 pCi/cm^3 in air or 1 pCi/cm^2 on ground surfaces. Since the models used in RADRISK are linear, these results may be scaled to evaluate other exposure conditions. The cohort consists of an initial population of 100,000 persons, all of whom are simultaneously liveborn. In the scenario employed here, the radiation exposure is assumed to begin at birth and continue throughout the entire lifetime of each individual.

No member of the cohort lives more than 110 years. The span from 0 to 110 years is divided into nine age intervals, and dose rates to specified organs at the midpoints of the age intervals are used as estimates of the annual dose during the age interval. For a given organ, the incremental probability of death due to radiation-induced cancer is estimated for each year using radiation risk factors and the calculated doses during that year and relevant preceding years. The incremental probabilities of death are used in conjunction with the actuarial life tables to estimate the incremental number of radiation-induced deaths each year.

The estimation of the number of premature deaths proceeds in the following manner. At the beginning of each year, m, there is a probability P^N of dying during that year from nonradiological causes, as calculated from the life table data, and an estimated incremental probability P^R of dying during that year due to radiation-induced cancer of the given organ. In general, for the m-th year, the calculations are:

M(m) = total number of deaths in cohort during year m,
= $[P^N(m) + P^R(m)] \times N(m)$

Q(m) = incremental number of deaths during year m due to radiation-induced cancer of a given organ,
= $P^R(m) \times N(m)$

$N(m+1)$ = number of survivors at the beginning of year $m + 1$
= $N(m) - M(m)$
$(N(1)=100{,}000)$.

P^R is assumed to be small relative to P^N, an assumption which is reasonable only for low-level exposures (Bu81), such as those considered here. The total number of incremental deaths for the cohort is then obtained by summing $Q(m)$ over all organs for 110 years.

In addition to providing an estimate of the incremental number of deaths, the life table methodology can be used to estimate the total number of years of life lost to those dying of radiation-induced cancer, the average number of years of life lost per incremental mortality, and the decrease in the population's life expectancy. The total number of years of life lost to those dying of radiation-induced cancer is computed as the difference between the total number of years of life lived by the cohort assuming no incremental radiation risk, and the total number of years of life lived by the same cohort assuming the incremental risk from radiation. The decrease in the population's life expectancy can be calculated as the total years of life lost divided by the original cohort size $(N(1)=100{,}000)$.

Either absolute or relative risk factors can be used. Absolute risk factors, given in terms of deaths per unit dose, are based on the assumption that there is some absolute number of deaths in a population exposed at a given age per unit of dose. Relative risk factors, the percentage increase in the ambient cancer death rate per unit dose, are based on the assumption that the annual rate of radiation-induced excess cancer deaths, due to a specific type of cancer, is proportional to the ambient rate of occurrence of fatal cancers of that type. Either the absolute or the relative risk factor is assumed to apply uniformly during a plateau period, beginning at the end of the latent period.

The estimates of incremental deaths in the cohort from chronic exposure are identically those which are obtained if a corresponding stationary population (i.e., a population in which equal numbers of persons are born and die in each year) is subjected to an acute radiation dose of the same magnitude. Since the total persons years lived by the cohort in this study is approximately 7.07 million, the estimates of incremental mortality in the cohort from chronic irradiation also apply to a one year dose of the same magnitude to a population of this size, age distribution, and age-specific mortality rates. More precise life table estimates for a specific population can be obtained by altering the structure of the cohort to reflect the age distribution of a particular population at risk.

B.3 Risk Analysis Methodology

Risk estimates in current use at EPA are based on the 1980 report (BEIR-3) of the National Academy of Sciences Advisory Committee on the Biological Effects of Ionizing Radiation (NAS80). The form of these risk estimates is, to some extent, dictated by practical considerations, e.g., a desire to limit the number of cases which must be processed for each environmental analysis and a need to conform to limitations of the computer codes in use. For example, rather than analyze male and female populations separately, the risk estimates have been merged for use with the general population; rather than perform both an absolute and a relative risk calculation, average values have been used.

The derivation of the risk estimates from the BEIR-3 report is presented in Chapter 8. A brief outline of the general procedure is summarized below. Tables referenced from Chapter V of NAS80 are designated by a V prefix.

(1) The total number of premature cancer fatalities from lifetime exposure to 1 rad per year of low LET radiation is constrained to be equal to the arithmetic average (280 per million person rad) of the absolute and relative risk values (158 and 403) given in Table V-25 of the BEIR-3 report (NAS80) for the L-L and $\overline{\text{L-L}}$ models for leukemia and solid cancers respectively.

(2) For cancers other than leukemia and bone cancer, the age and sex specific incidence estimates given in Table V-14 were multiplied by the mortality/incidence ratios of Table V-15 and processed through the life table code at constant, lifetime dose rates of 1 rad per year. The resulting deaths are averaged, using the male/female birth ratio, and proportioned for deaths due to cancer in a specific organ as described in Chapter 8. These proportional risks are then used to allocate the organ risks among the (235.5) deaths per million person rad remaining after the 44.5 leukemia and bone cancer fatalities (Table V-17) are subtracted from the arithmetic average of 280 given in Table V-25.

(3) The RADRISK code calculates dose rates for high- and low-LET radiations independently. A quality factor of 20 has been applied to all alpha doses (ICRP77) to obtain the organ dose equivalent rates in rem per year. The derivation of the proportional organ risks and mortality coefficients for alpha particles are, however, based on the dose in rad as described in Chapter 8, Table 8-6.

A typical environmental analysis requires that a large number of radionuclides and multiple exposure modes be considered. The RADRISK code has been used to obtain estimates of cancer risk for intakes of approximately 200 radionuclides and external exposures by approximately 500 radionuclides. For each radionuclide and exposure mode we assume that each member of a cohort of 100,000 persons is exposed to a constant radionuclide intake of 1 pCi/year, or a concentraton of 1 pCi/cc-year for air immersion, or of 1 pCi/cm^2-year from the ground surface, until they die or are 110 years old, the maximum cohort. The mean life span of the cohort population is 70.7 years, a result obtained from 1970 age specific mortality rates. The calculated dose rates and mortality coefficients described in the preceding sections are then processed through the life table subroutine of the RADRISK code to obtain lifetime risk estimates. At the low levels of contamination normally encountered in the environment, the life table population is not appreciably perturbed by the excess radiation deaths calculated and, since both the dose and risk models are linear, these unit exposure results may be scaled to reflect excess cancers due to the radionuclide concentrations predicted in the analysis of a specific source.

As noted in the discussion of the life table analysis, risk estimates for chronic irradiation of the cohort may also be applied to a stationary population having the same age-specific mortality rates as the 1970 U.S. population. That is, since the stationary population is formed by superposition of all age groups in the cohort, each age group corresponds to a segment of the stationary population with the total population equal to the sum of all the age groups. Therefore, the number of excess fatal cancers calculated for lifetime exposure of the cohort at a constant dose rate would be numerically equal to that calculated for the stationary population exposed to an annual dose of the same magnitude. Thus, the risk estimates may be reported as a lifetime risk (the cohort interpretation) or as the risk ensuing from an annual exposure to the stationary population. This equivalence is particularly useful in analyzing acute population exposures. For example, estimates for a stationary population exposed to annual doses which vary from year to year may be obtained by summing the results of a series of cohort calculations at various annual dose rates.

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